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"WORD-BLINDNESS" IN SCHOOL CHILDREN *

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The material included in this preliminary report has come from several sources but was assembled chiefly during an experimental clinic held by members of the Iowa State Psychopathic Hospital Staff in Greene County, Iowa, in January, 1925. Among those children who were reported to the clinic by their teachers as "dull, subnormal, or failing or retarded in school work" was a fairly high proportion whose chief difficulty was in learning to read. Two of these would fit Hinshelwood's criteria of true "congenital word-blindness," and one of these two cases (M. P.) also gave bizarre written productions.

Because of his striking disability, M. P. was admitted to the State Psychopathic Hospital, and his case was there studied more thoroughly than was possible in the clinic. The results of this study are here reported in full.

Observations suggesting an explanation of one of the factors in this case are recorded from other cases. These were the presence of mirror reading, mirror writing and a strong tendency to attempt to read parts or all of a word from right to left and confusion of those letters in which orientation is essential.

Only a few of the studies from the literature of mirror writing and left-handedness have been reviewed in preparing this preliminary report, but these indicate the great frequency of these conditions in "defective" children. The method of writing in alternate directions and with the letters correspondingly oriented as seen in certain ancient documents indicates that our present method of dextrad writing with single orientation of letters has been arbitrarily fixed by custom.

The views on "congenital word-blindness" in the medical literature seem untenable as an explanation of these cases. An hypothesis more in harmony with present conceptions of the aphasias and based on the structural relations and the probable physiologic activities within the visual cortices of the two hemispheres is offered, and a new, descriptive term is suggested for this group.

Some points of psychiatric import are suggested. Certain reactions to the disability might readily serve to establish determining character

* From the laboratories of the State Psychopathic Hospital, Iowa City.

* Read at the Fifty-First Annual Meeting of the American Neurological Association, Washington, D. C., May, 1925.

traits. Moreover, it seems probable that psychometric tests as ordinarily employed give an entirely erroneous and unfair estimate of the intellectual capacity of these children.

The implications from the standpoint of education are challenging. If the views herein expressed are proved to be correct by further observation and experiment, there is reason to believe that the majority of these disabilities can be entirely overcome by special training. The methods of training, however, must be developed in consonance with the neurologic background and tested by carefully controlled experiment.

SOURCE OF AUTHOR'S MATERIAL

During two weeks in January, 1925, a mobile mental clinic [1] was held as an experiment in Greene County, Iowa, by a group of members of the staff of the State Psychopathic Hospital, consisting of the director of the hospital acting as psychiatrist, the chief social worker of the hospital, a graduate student assistant in social work, the hospital psychologist and a graduate student assistant in psychology.

Cases were referred to this clinic by four agencies in the county—the physicians, the county attorney, the secretary of the social service league and the schools. An announcement was made to the school teachers that the clinic would be ready to study pupils who seemed unusually bright, and in whom the question of double promotion or enlargement in scope of the school work might be indicated, pupils who presented behavior problems and those who were considered defective or who were retarded or failing in their school work.

This last group, the retarded, the failures and those considered by their teachers to be defective, proved to be an extremely interesting selection. Altogether 142 pupils of the grade and high schools of the county were studied. Eighty-eight of the 142 fell in the group referred by the teachers as deficient. Psychometric ratings were made of eighty-four of these, but they did not agree closely with the teachers' estimates of the children's abilities.

The distribution of this group by psychometric ratings obtained by individual Stanford-Binet tests was as follows:

TABLE 1.—*Psychometric Ratings of Eighty-Four Deficient Students*

Very superior intelligence, Stanford-Binet Test, intelligence quotient, 120 or over.	1
Superior intelligence, Stanford-Binet Test, intelligence quotient, 110 to 119	0
Average intelligence, Stanford-Binet Test, intelligence quotient, 90 to 109	31
Dull normal intelligence, Stanford-Binet Test, intelligence quotient, 80 to 89	20
Marginal defective, Stanford-Binet Test, intelligence, 70 to 79	18
Moron, Stanford-Binet Test, intelligence quotient, 50 to 69	13
Imbecile, Stanford-Binet Test, intelligence quotient, 25 to 49	1
	84

1. To be reported in greater detail elsewhere.

It is obvious from this distribution that other factors than mental defect were largely responsible for the poor work of these children. Among these factors appeared a special difficulty in learning to read. Fourteen of these eighty-eight children were reported by the teachers as having great difficulty in learning to read, or were seen by their grades or by cursory examinations in the clinic to have some special limitations here. One other student referred by his teachers as "nervous" proved to be of this type. Because the method of selection did not specifically call for those with reading disabilities, it is highly probable that this number does not adequately present the whole problem in these schools.

The distribution by grades and intelligence quotients of the fifteen who were recognized as having this disability in greater or less degree was as follows:

TABLE 2.—*Distribution by Grades and Intelligence Quotient of Students with Difficulty in Learning to Read*

School Grade	Case	Stanford-Binet Intelligence Quotient
First	Merle	103
Second	Dale	91
Second	Francis	122
Second	Douglas	92
Third	Clarke	102
Third	Ludlow	105
Third	Etta	91
Third	Wayne	96
Third	Karl	99
Third	George	75
Third	Derald	102
Seventh	Donald	70
Eighth	John	72
Ninth	Jack	85
Ninth	M. P.	71

The two outstanding cases were those of boys about 16 years of age, who had reached the ninth grade, and the degree of their disability was so extreme as to warrant their inclusion in the group of cases described by Hinshelwood[2] under the name of congenital word-blindness. In addition to a practically complete inability to read, one of these two boys (M. P.) had submitted some extremely curious productions as written exercises in school.

STUDY OF A TYPICAL CASE

During the clinic, M. P. was tested by the Stanford-Binet method and showed the following rating: Age, 16 years, 2 months; mental

2. Hinshelwood, J.: Congenital Word-Blindness, London, H. K. Lewis, 1917.

age, 11 years, 4 months; intelligence quotient, 71. During the psychiatric examination which followed, however, I was strongly impressed with the feeling that this estimate did not do justice to the boy's mental equipment, and that the low rating was to be explained by the fact that the test is inadequate to gage the equipment in a case of such a special disability. Further, it was easily seen that while he was unable to recall the visual impressions of words clearly enough to recognize them in print, he did make facile use of visual imagery of objects of rather complex type. I asked him, for example, questions concerning the

I. Conter in 1777.

The Enlgand camele stosent in cane hosterson the their last scoune and honster posterson and roscson and sean 1000 scouter then to pantore and the fosteron the secounter tall theen heuster and at dog befor mostir hasden or Miss Hesent well be a wholl and the stosert heute Capuster and Husglas suerist to Austerson. Scallson stosert of Kronton and uester to called stosert to counter and cane to the scoperson cane doses the toster of the coster of the Boster and tater to last scastorn and to nicast to. But the Anson custome stosent tae the the cashie so the peoper sconer tabe at his the usenter eacet.

Dealess of faster to the Last of the scomet of caperson tall the and there can to last a burst Uster and heust Lester and cane to the art last.

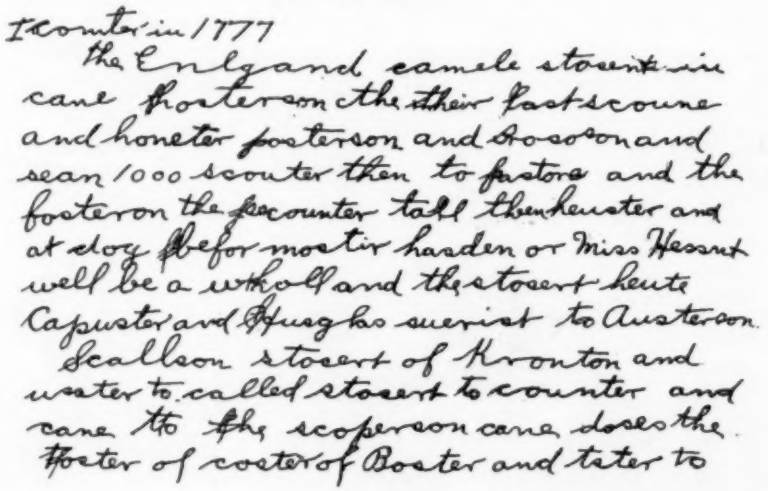


Fig. 1.

adjustment of bearings in the V type automobile engine which required a good visualizing power for answer, and his replies were prompt and keen.

Figure 1 gives above, a transcript of a page of one of his written productions, and below, a part of it in fac-simile. This was submitted as a composition in American History describing the English Campaign of 1777.

The challenge to interpretation contained in this production, together with the doubts of the accuracy of his mental rating and the general interest of the reading defect, led to his admission to the psychopathic hospital for more extended study and experiment.

Additional tests given in the psychologic laboratory of the hospital gave the following results: The Stanford-Binet test was repeated, but certain alternative tests were substituted; others, when permissible, were given orally, and he was tried by tests higher in the scale. This resulted in a mental age of 13 years, 10 months, and an intelligence quotient of 86—fifteen points higher than in the initial test, thus placing him in the dull normal instead of the marginal defective group. He still gave the impression, however, to one who had learned to estimate mental defect before the widespread use of mental tests, of a much better equipment than even this second rating indicated. By the Pintner-Patterson performance tests, his accomplishment was satisfactory for adults. On twelve of the twenty-two items he earned maximum scores. By the Healy pictorial completion test No. 2 his score was 90 out of a possible 100, which is a superior performance for adults. By the Stenquist mechanical assembly test, No. 1, he earned a score of 82, which would place him on a level with the highest 1 per cent. of unselected army draft recruits. He solved the Freeman mechanical puzzle box in 102 seconds on the first trial and 72 seconds on the second, which is a superior performance. Tests with Nagel's color blindness cards gave no evidence of defect in color vision. His capacity to read in a mirror was tested, and it was found that this reversed presentation made no difference in his ease of reading.

As the next step in the study of his case, he was tried repeatedly by the series of tests devised by Head [3] in his studies of aphasia. The tests of capacity to draw and of the mimeokinetic functions with and without the mirror were tried briefly. The others, however, were carried out repeatedly, and as a result it was obvious that there was no difficulty in the recognition of objects or in calling them by name when visually presented, or in indicating an object by pointing when its name was called. He recognized all of the letters of the alphabet and the arabic numerals promptly, and called each by name correctly without hesitation. When names of objects were visually presented, however, there was prompt and correct response in only a few instances, and these usually only in the case of the simplest words. With the longer and less simple words there was delay with evident effort to spell out the word and vocal or subvocal attempts at pronunciation of its letters and syllables, with varying degrees of success. This presentation of words was carried out in three forms: in script, in typewritten words and by means of black paper letters one quarter of an inch high mounted on white cards. There was no great difference in the ease of reading of any of these, although the gummed letters on cards seemed a trifle easier for him.

3. Head, Henry: Brain **43**:2, 1920.

He was then tested for his capacity to copy in script from printed text with the result shown in Figure 2.

It will be noted that while there is a certain immaturity about the writing, it is by no means a bad production for a 16 year old boy, and that the copy is practically letter perfect. When asked, however, for the content of what he had just copied, he replied: "I don't know, I didn't read it." He was then asked to read it from his own handwriting, and his pronunciation, reproduced as accurately as I could record it, is shown at the bottom of Figure 2. He produced this slowly and with obvious effort, and in the case of most of the words by sounding out part or all of the individual letters before attempting to pronounce the word as a whole. The similarity of syllabic structure of some of these

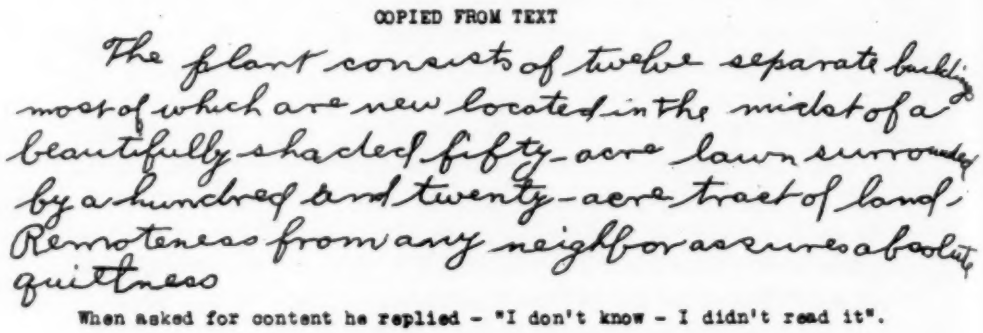


Fig. 2.

words to that of the neographisms in his original composition (Fig. 1) is apparent. He was then asked to read the same material from the original printed text, and while his errors were not quite the same, the general character was quite like that from his own copy of it.

A copy of a children's edition of "Aladdin and the Wonderful Lamp" was then given him with instructions to read the first page aloud. His efforts at this are shown in Figure 3. The text is given at the top of the illustration; next is my record of his reading. He was then asked to write the same material from dictation (given two or three words at a time), and a copy of this production forms the third part of the illustration. After he had attempted to read this material and then had written it to dictation, he was asked to reproduce its contents from memory. The result is recorded at the bottom of the illustration. When questioned as to his inability to remember the

material, he replied: "It takes me so long to spell out some words that by the time I read them I forget what was ahead of them."

To test his auditory memory the second page of "Aladdin and the Wonderful Lamp" was read aloud to him, and he was instructed to listen and be prepared to repeat the story. The text was:

"How would you like to work for me, my boy? I will pay you well," he said to Aladdin. Aladdin said nothing would please him more if his mother would let him.

The poor widow was overjoyed to have Aladdin make some money, and he started next day to work for the magician who treated Aladdin very kindly, giving him lots of fine clothes and paying him well.

Everything went well until one day the magician said to Aladdin: "Come, my boy, I will take you for a walk and show you some very fine things."

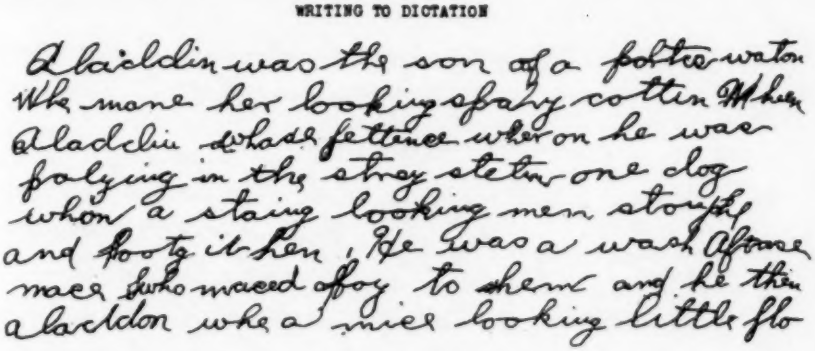
TEXT

Aladdin was the son of a poor widow who made her living spinning cotton. When Aladdin was fifteen years old he was playing in the street one day when a strange-looking man stopped and looked at him. He was a wicked African magician who needed a boy to help him, and he thought Aladdin was a nice looking little fellow.

READING (Time 6' 10")

Aladdin was the son of a poor woman who made her livin spinning cotton. When laddin was fighting were old he was playing in the street one day when a standar looking man stepped up and looked at him. He was a wicked African macrican who nidded - needed a boy to help him and he took Aladdin with a nike looking little flume.

WRITING TO DICTATION



REPRODUCTION OF ABOVE AFTER READING AND WRITING

There was Aladdin. His mother took in wash - I mean - knitted wool - I mean cotton and he was playing in the street one day and a cross African man rode up and he thought he was good looking so he took him with him.

Fig. 3.

M. P.'s reproduction of this was:

He asked Aladdin if he would like to work for him and said he would pay him. Aladdin said he would if his mother would let him. His mother was glad to have him earn some more money and he went to work for the *musician* who bought him some clothes and paid him. Everything went alright until the *musician* asked him, "When will you take a walk *in the woods* with me and see some fine things?"

It will be seen that this is quite a creditable reproduction of the content of the presented text. Two errors appear—"magician" becomes "musician" and the phrase "in the woods" is inserted.

He was next asked to describe in his own words some of the events of the hospital day, and then to write them down. Figure 4 gives above his verbal description and below a fac-simile of his writing of the same material, and at the bottom, of his production on writing to dictation.

Several words were next dictated, first as words and then letter by letter, and a written record of them was requested. The whole words were better written than when spontaneously done, and when spelled out letter by letter slowly enough for him to write each letter before the next was heard, he wrote them perfectly. When, however, all the letters of a word were verbally given and he was instructed to wait until the

VERBAL DESCRIPTION

We played volley ball today. The way to play it is this - put up the net and knock the ball over the net and the boy on the other side knocks it back and if you can not there is a point for him.

WRITING ABOVE

We perple volleg ball to clay the way to perple it is this put up the net and knock the ball over the net and the boys on the other shide knock it back and if you can not there is a port for he.

WRITTEN TO DICTATION

We play valley ball to clog the awaye to play it is this put over the net and knock ball over the net and boys of the other shide knock it back and if ybu can not there is a port for he.

Fig. 4.

word was spelled completely before beginning to write, his attempts at writing were again full of errors, suggesting an extremely short memory span for series of letter sounds. His span for numerals in other tests was variable, but much better than that indicated for letters.

A large series of separate words made by gummed letters on cards was then prepared, and his ability to read these was tested. Many of these formed series of similar words, such as: mare, fare, bare; mend, rend, tend; lack, back, hack; etc. Others were longer common words, like tonight, today, tomorrow, and some were two-syllable words in a series, like target, tarnish, tardy, etc. These two-syllable words were presented as units and also separated into syllables. Altogether, about

250 cards were used in various ways. Reading of two-syllable words was definitely easier when the syllables were separated, and some words which were impossible for him to read as a whole could be made plain for him by building them letter by letter, using letters mounted on blocks (anagrams). In going over the same series of words day after day, some distinct facilitation was observed, and yet this was irregular, and often words remembered from one day to the next were missed when re-presented on the third day. In one series of sixty-nine simple words, none with more than two syllables, after taking all the time desired for spelling out and corrections, he read only thirty-two correctly.

Recognition of the letters of the alphabet was immediate always, and the name of each letter was correctly and promptly given. When, however, he was asked to sound out the letters as presented, there was evidence of a striking lack of association of certain letter sounds with the corresponding letter form. All of these sounds were possible for him, and were consistently used in speech, but when a certain printed vowel was presented to him, usually only one of its varied sounds came as a spontaneous response. When pressed for other possible sounds, after considerable hesitation, he usually would offer another; but some of the common sounds of vowels were never produced as a response to visual presentation of the corresponding letters standing alone, although in short words which he could read at sight, he often used all the sounds of each vowel. In the first column of the following table are shown the letters visually presented; in the second, the sound response immediately offered; in the third, the response in answer to the question as to whether this letter might have other sounds, and in the fourth, the common sounds of the letters which were never produced as a spontaneous or requested response to the letter standing alone.

TABLE 3.—*Letter Test*

Letters	When Read	When Questioned	Not Produced
a	ă	ĭ	ā ä*
e	ĭ	ē*	ĕ*
i	ĭ	ī (slow)	.....
o	oo	.....	ō*
u	yŭ	ŭ	ū
y	wŭ	wĭ	ĭ* ī
w	wŭ	.....	.....
c	k	s (rare)	.....

* These sounds were frequently reproduced in short words which he could read at sight, such as me, bet, my, old, farm, etc.

The effect on his reading of this faulty range of sound associations with certain letters is seen in Table 3.

TABLE 4.—*Word Test*

Word Presented	Read as
Child	Chĭlled
Chilly	Chĭlwŭ
Twenty	Twĭntwŭ or twentwŭ
Blue	Blwŭ
Ball	Bĕll, bĭll (various times)
Tend	Tĭnd
Dug	Dwŭg
Check	Chŭck
Chock	Chŭck
Bend	Bĭnd
Nice	Nĭke

The second noted element was a tendency to reverse parts or all of a word and read the letters from right to left. A few examples of this follow:

TABLE 5.—*Word Test*

Word Presented	Read as
Dug	Gud
Gray	Gary
Tar-nish	Tar-shin
Pardner	Ponder
Mend	Medn
Fend	Fedn
Tomorrow	Tworrom

Both of these factors may be seen operating together in such pronunciations as: twĭvl for twelve, rĭdn for rend, tĕgrĕt for target, blĕwŭ for blue, etc.

A considerable series of three letter nonsense syllable was also presented by means of the anagrams and gummed letters. In this experiment he was told not to try to find a word with meaning but simply to sound the letter groups as they looked to him, and here again the two elements in his disability mentioned above became evident. Again when trying to read there was a striking tendency to get the sound of a few of the initial letters and then to "jump" at the rest. Blue was frequently read as black or blow. When check was presented, he first read it as chick. Chick was then presented and correctly read, but when check was again presented it was read first as cheek, then chuck, then choke. This tendency was much more pronounced after the tests had been under way for a short time, and apparently increased with fatigue.

In his attempts at writing there was also frequent elision of letters resulting in phonic simplifications, which were frequently readily understandable although bizarre in appearance. Work-up became wrkp, supper became supr, dining-room became dierom, cargo became crgo, etc. Several grades of error in writing were observed. Some might

rank as extreme instances of misspelling, as blou for blue, weit for white, blak for black, etc. Others show the substitution of other words with similar initial letters, as people for play, port for point, looking for living, etc. Still others show distortions which have almost or entirely lost their sound relation, as hlo for yellow, gen or green, waton for widow, spary for spinning, feltence for fifteen, etc. The number of neographisms in any written production seemed to be in indirect relation to the degree of effort expended, and hence to the speed of production. When M. P. was urged to take the time he needed and spell out each word, the product was very much better than when he was told to write rapidly, but some errors crept in even in his most careful work, and such productions were an exceedingly slow and arduous task. Fatigue effects appeared early in these efforts, and apparently served to increase the production of bizarre words. There is a striking tendency in his composition (Fig. 1) to repeat new words of almost similar form, such as hosterson, rososon, fosteron, scoperson and toster, coster, Boster, tster, etc. There was also in his reading of a series of words from cards a noticeable perseveration of letters. Bend was called bĭnd, and when followed by lend, the latter became blĭnd; rend was called rĕdn, and when followed by send, the latter became srĕdn, etc. This tendency was never observed when the first word was one which he knew and recognized at sight, so that it seemed as though the unsolved puzzle of a word which did not bring its proper sound association continued its influence on the next presented. The striking neographisms which characterize Figure 1 can probably never be interpreted, as M. P. himself has forgotten the details of the campaign which he had in mind when this was written.

A few brief experiments were tried in training M. P. to learn to recognize some of the simple words that proved hard for him. Twenty was one of these, although he read it promptly as twenty if spelled t-w-a-n-t-i. Twe was presented, and he pronounced it twē. When urged to try another sound for the e, it was produced as twĭ. The twĕ sounds were then pronounced for him, and twen was presented and read as twēn. On urging, it was finally produced as twĕn, and then twent was offered. This was pronounced as twĕnt, but when the y was added it was pronounced as twentwŭ. Y was then pronounced for him as ĭ, and he was drilled with ly, ty, and ry, etc.; and again twenty was presented, and this time correctly pronounced. This exercise was exceedingly slow—it required eighteen minutes, ten seconds, and while the lesson was retained for one day, on the next it had been forgotten, and twenty was again twentwŭ.

Our studies of M. P. show that he made active use of visual images of objects as, for example, in his work with the Healy pictorial completion test. His capacity to copy indicated that his visual equipment

was adequate to receive correct impressions of the stimuli and to translate them into one form of motion-copying. He could translate his ideas into speech, and his product here must be considered quite good when allowance is made for the fact that practically his whole vocabulary had been acquired by hearing. He could not, however, translate these same ideas into writing. It is true that with great effort, much expenditure of time and many errors, he could make out many words by reading and could write many correctly, but prompt and facile reading and writing were impossible for him. From his writing to dictation, it is clear that the sounds of dictated words did not arouse accurate visual images of letters, except when he laboriously spelled out each word a letter at a time, and even then many were incorrect. His composition suggests that his ideas were even less effective than were dictated words in bringing up the proper visual associations. Conversely, the printed or written word failed to arouse its corresponding concept or its auditory association, so that silent reading as well as reading aloud was impossible for him. Emphasis should be laid, however, on the point that it was in dealing with visual symbols *only* that this associative difficulty occurred. As our records by the Head tests show, there was no interference with the immediate and correct association of objects with their auditory symbols or of the auditory symbol with the corresponding object. He recognized objects and called them by name promtly and correctly, and also indicated them correctly when the name was called. This analysis would seem to indicate a practically complete failure of association operating electively at the symbolic level between the strictly visual cortices and the great association area, as revealed in his difficulty in reading and, conversely, between the association sphere and the visual areas, as shown by his attempts at writing spontaneously and to dictation.

COMPARISON WITH OTHER CASES

The other fourteen cases of reading difficulty encountered in the clinic were necessarily more hastily reviewed, but from the data obtained from these and a few additional cases seen in the outpatient department and elsewhere, it seems that the tendency to read from right to left. leading to confusion of such words as on and no or not and ton and the tendency to reversals leading to difficulty in telling a lower case p from q or b from d is of constant occurrence in these children, and tends to confirm the findings in M. P.'s case.

Some of these subjects were tested with a mirror, and were found to read as readily or even more so when the text was seen in mirror image than when seen direct. The motor facility with each hand both in ordinary writing and in mirror writing was also tested briefly in several

cases, and some were found who could write practically as well with one hand as with the other. Others showed varying degrees of success in producing mirror writing. Two wrote their names not only in mirrored reversion but upside down as well, and one boy was found who could read at least as easily, if not more so, when the text was upside down and also seen in a mirror. One child, Clarke C., aged 10, with an intelligence quotient of 102, made four mistakes in simple words (white, bend, nod, dance) in reading from the printed text directly, but he read the whole sentence of fifteen words promptly and correctly when seen in the mirror. This boy had found the second grade in school very hard, was repeating the third grade and was reported by his teachers as "naturally dull." Except in his reading, however, he gave the impression of an alert and interested boy, though very slow and insecure in his motor reactions. His own description of his difficulty is worth quoting. "Hardest one (study) is reading. Arithmetic has always been my easiest study. Mother says there is something funny about me because you could read anything to me and I'd git it

Fig. 5.

right away, but if I read it myself I couldn't git it." This aptly characterizes the group. Another, John C., aged 17, with an intelligence quotient of 72, showed a striking insecurity in telling lower case p from q and b from d. These letters were mixed both when seen directly and in the mirror, and there was no consistency in his mistakes —in one word b would be correctly read, while in the next it would be read as d. John formerly stuttered badly, and still shows traces of this difficulty. He throws and writes with his right hand, but handles a shovel and pitchfork as a left-handed person would. His father is left-handed in everything but writing. Jack D., a 16 year old boy in the ninth grade, gave a Stanford-Binet intelligence quotient of 85, which probably does not represent his full capacity, as he has always suffered from a marked reading disability. He showed an additional factor in that b and d were not only confused with each other, but also with p and q as though there were a factor of inversion here as well as a mirrored reversal. When asked to attempt to write his name in mirror writing after having seen a pattern so written by the observer, he wrote it as seen in Figure 5.

His attention was called to the fact that this was upside down, but not a mirror image, and he was asked to try again with his left hand, with the result shown in Figure 6.

As will be seen, the first is inverted, the second reversed.

This inversion in writing occurred in partial form in one other boy, Karl K., aged 9, third grade, intelligence quotient, 99. Copies of his direct writing with his right hand and his attempts at mirror writing

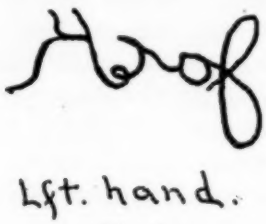


Fig. 6.

are shown in Figure 7. In the attempts at mirror writing, he has produced with each hand a mirrored form of Ka, but has merely inverted the rl.

Karl also apparently had difficulty in both vertical and lateral orientations in reading, as shown by the fact that lower case p was often confused with b and d, although q was consistently recognized correctly.

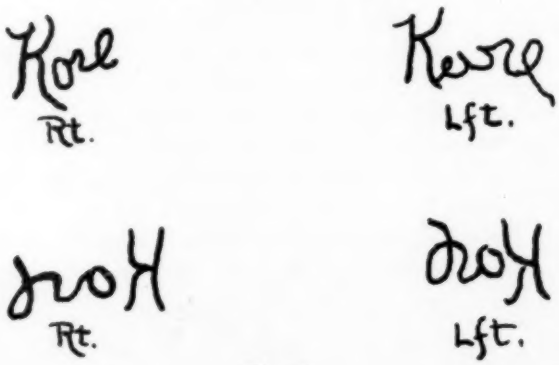


Fig. 7.

M. R., aged 30, intelligence quotient, 93, came to the outpatient department of the hospital for vocational advice. She had always had difficulty in spelling and in learning things by reading, although she had managed to graduate from college by dint of hard work and possibly some favoring by her teachers because of brighter brothers and sisters who had preceded her. She gave interesting evidence on brief study of the tendency to reversal of direction in reading. She had never attempted to read with a mirror, but on the first trial she read the first

five simple words of a printed sentence, the sixth word was "round," and as this was not immediately recognized, she started to spell it audibly as b-n and then stopped as though puzzled. Apparently as long as the words were short and easily read as a whole there was no difficulty with the mirrored images, but when one was met that did not immediately arouse its proper auditory association, she reversed it and began to try to puzzle it out by spelling it backward, suggesting a return to an earlier habit of approach to difficult words.

One of the most convincing observations came from a case, M. O., which was not one of the clinic series. This was a little girl who on her first attempts at writing produced a definite mirrored reversal of each letter with her right hand. It seems axiomatic that the visual image of the letter that was called up by its name, and which must have served as the motor pattern, existed in her brain as the mirrored reversal of that which we consider the correct form of that letter.

Certain additional facts of interest, related to the general problem of cerebral dominance, came out in this group. Many of these children are clumsy with both hands, or had been so in earlier childhood. They are often of the "motor incoordinate" type with evidences of mild apraxia. Some of them give a history of delay in learning to talk and walk and of lack of nicety of balance and consequent frequent falls and of indecision in the choice of the right or left hand in using the knife, fork and spoon, all of which speak for a definite delay in decisive dominant control of the motor mechanisms. Again, several authors have called attention to the frequent occurrence of stuttering in left-handed children and in those with no established lead, when learning to write with the right hand, and, in some, of the rapid disappearance of this trouble when writing with the left hand is permitted. In our series of fifteen cases of reading disability seen in the Greene County Schools fourteen were boys—a fact of interest in connection with the much greater incidence of stuttering in boys than in girls, and of these fourteen boys there were three who stuttered or who had formerly done so and four others whose speech had a peculiar labored hesitancy quite like that of one who has been broken of stuttering.

LEFT-HANDEDNESS AND MIRROR WRITING IN DEFECTIVE CHILDREN

No attempt has been made in preparing this preliminary report to go into the literature of mirror writing and allied phenomena exhaustively, but a few facts of interest to this study have been encountered. Gordon [4] reports the finding of an extremely large proportion of left-handed children and mirror writers among the pupils of the special

4. Gordon, Hugh: Brain **43**:4, 1920.

schools for "defectives" in London and Middlesex. He explains that these schools are not institutions for the feebleminded but are provided for children "who not being imbecile and not being merely dull or backward are defective, that is to say, children who by reason of mental or physical defect are incapable of receiving proper benefit from the instruction in the ordinary public elementary schools but are not incapable by reason of such defect of receiving benefit from instruction in special classes or schools." It will be seen therefore that he was dealing with children of the "ungraded" or "special" class type rather than defectives as we use the term. Gordon's first observation was that there was a much higher percentage of left-handed children in one of these special schools than in the ordinary elementary schools. He then carried out a large series of tests to determine motor predilection for right or left, and found that in schools for the defective the left-handed boys constituted 16.6 per cent., the girls 20.7 per cent., or an average of 18.7 per cent., which he states is more than two and a half times as high as in the ordinary elementary schools. He quotes the proportion of left-handedness in normal persons as found by Ogle as 4.5 per cent., Gould 6 per cent., Jones 4 per cent., Malgaigne 8 to 10 per cent., Hecht and Langstein 12 per cent. and by Masini as 10 per cent. It will be seen that his findings in the "defective" children are considerably above the highest of these figures. Gordon also studied mirror writing in these children, and found that 8 per cent. of a total of 1,350 were mirror writers. The product was very good in 56 per cent. of these; good in 29 per cent.; fair in 7.5 per cent. and bad in 7.5 per cent. Fifty-seven per cent. started to write at the right margin of the paper, 13 per cent. in the middle and 30 per cent. at the left margin. He quotes an interesting remark by the head teacher of one of these schools, who stated that he had frequently noticed a great improvement in a child's intelligence and school work, with a natural change of left to right hand. Gordon offers the hypothesis that something has occurred which has interfered with the proper functioning of the dominant hemisphere.

Fildes [5] has made an extended study of mirror writing. She reports that mirror writing is common among young children and among defective children and varies from an occasional reversal of single letters to complete reversal of all letters and words (true mirror writing), which is comparatively rare. It is found most frequently among the left-handed, but also occurs among right-handed children who have never written except with the right hand. She carried out an extended series of experiments with mirror writers as a result of which she suggests that the main cause for continued error among the defectives is their tendency to repeat a mistake once made in spite of correction. She

5. Fildes, Lucy G.: Brit. J. Psychol. **14**:24, 1923.

does not, however, offer any hypothesis to explain this tendency, and apparently has not considered the possibility of a mirrored sensory image as the source of this persistence. As a further cause, she hypothecates poor initial observation. That this factor may play some part seems obvious, but that it should be a major factor, is out of harmony with the active use of visual images of objects which many of these children show. M. P.'s score, for example, in the Healy pictorial completion test indicates a high degree of keen visual observation. In conclusion, Fildes reports that a tendency to reversal existed in greater or less degree in all subjects tested, both normal and defective, and may therefore be regarded as a common one.

That mirror writing with the left hand is the normal sinistral expression has frequently been recognized. Javal [6] emphasizes that the complete reversal of direction and orientation of letters is the normal writing for the left-handed and advises that if a patient with a right-sided paralysis or cramp wishes to acquire left-hand writing as rapidly as possible, he should write in mirror fashion. He cites the mirror writing in the manuscripts of Leonardo da Vinci. An interesting account of da Vinci with reproductions in fac-simile of some of his mirror writings was published by Caetani [7] in the *Scientific Monthly*.

Prof. B. L. Ullmann of the State University of Iowa, in a personal communication, has called my attention to some suggestive facts from old Phoenecian, Greek and Latin inscriptions. The Phoenecian writing, like the Hebrew, ran from right to left and the letter K—kaph—in their inscriptions is reversed and appears as ⋊. When taken into the Greek, however, which was written from left to right, it became kappa and took the direction of our own K. An exactly parallel situation is found in the contrast between the Umbrian and Oscan Italic dialects which followed a sinistrad direction, and in which the K, S, E and other unsymmetrical letters are written in the sinistral form and the Latin which was a dextrad writing and used a letter orientation like our own. In many of the earliest inscriptions there was apparently great elasticity in orientation so that in some the same letter, such as an E for example, appears in both directions in the same inscription. Still more striking is the very old method of writing one line toward the right and then reversing and writing the next toward the left. This gave rise to a back and forth order across the page which was likened to the path of an ox in ploughing a field and hence has been described under the name of bustrophedon or ox turns. The "Old Forum Inscription," of which Huelson [8] says, "Among all the inscriptions

6. Javal: Physiology de la Lecture et l'Ecriture, Paris, 1906.

7. Caetani, Don Gelasio: The Scientific Monthly **15**:5, 1924.

8. Huelson, Charles: The Roman Forum, English trans. by J. B. Carter, New York, G. E. Stechert & Company, 1909.

(Latin) preserved on stone, it is certainly the oldest and is not later than the fifth century, B. C.," was a vertical bustrophedon in which the writing on the stone as erected goes from the top down on one line and from the bottom up on the next. The letters, however, are placed as though on a horizontal base line. Figure 8 is a tracing of Huelson's photographic illustration of this inscription on the four faces of the stone, shown in the horizontal position in order to be more easily legible.

As a whole, there is fair concurrence between the orientation of the letters and the direction of the line. The E, for example, faces con-

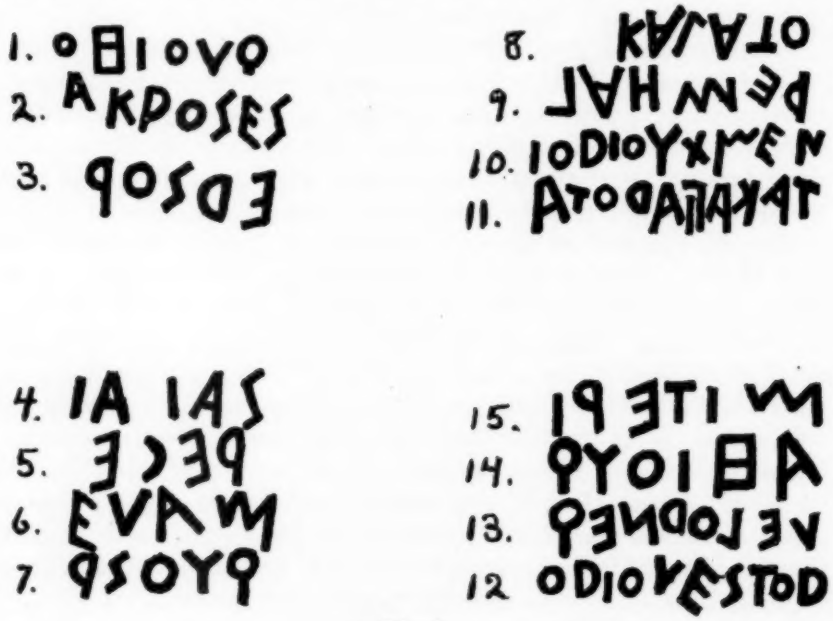


Fig. 8.

sistently in the direction of writing. With the A there is somewhat less certainty. In line 11, for example, the diagonal cross stroke appears in two different letters slanting up with the direction of travel and in two others slanting down. The S, however, has apparently become fixed in one orientation throughout the inscription. Lines 2, 4 and 12 are running to the right, and lines 3 and 7 to the left, but the S in all has the same form. Another interesting feature is shown by the complete inversion of lines 8 and 9, and from Huelson's numbering of 12 to 15 one would infer that these were also inverted with respect to the remainder. Professor Ullman further tells me that he has frequently encountered reversals of letter pairs in medieval manuscripts written by one scribe which are consistent enough to be considered characteristic of that individual writer.

Apparently, at one stage in the development of writing the orientation of individual letters was not of great importance, which probably means that the symbol could be read in either direction. The bustrophedon shows that at another stage both orientations were in use in recording and would imply that both left and right hand forms of letters assembled in corresponding order could be read by educated persons. The growing fixation of dextral orientation of unsymmetrical letters in dextrad writing as the languages developed would indicate that orientation of letters and direction of writing are intimately connected, but the bustrophedon would indicate that exclusively dextrad orientation is by no means determined by brain capacities, but has been established as a purely arbitrary limitation fixed by custom and education.

Sereni [9] calls attention to the fact that mirror writing with the left hand is an expression of the symmetry of build of the body. This is obvious when we consider that, so far as the motor mechanisms are concerned, any innervation of the muscles of the left hand will give a motion exactly opposite to that resulting from the comparable innervation applied on the right. Figure 9 shows these relations in graphic form. This again, however, does not take into account the sensory images which serve as the pattern for writing from memory. Writing in either the direct or mirrored form is possible to some people with either hand, and this potentiality is probably latent in us all though difficult to develop because of the exclusive training of one hand for writing. Sereni [9] reported the case of a man, aged 45, who, two years before, had suffered from a trauma to his right elbow, with a temporary loss of the use of his right hand. He learned to write in ordinary form with his left hand, but later he found that he could write in either the direct or mirrored form with either hand. The dextrad writing produced with the left hand was different from that produced with the right, but the mirrored left was like the dextrad right in its strokes, and vice versa. Sereni considers mirror writing as the carrying over, to one side, of a motor process acquired by the other.

Fildes and Myers [10] have reported a study of a 6 year old boy with a striking tendency toward mirrored reversal of letters and insecurity in recognizing the correct right-left orientation of many of them. He had several left-handed relatives. He had just begun to be taught to write with his right hand, but found difficulty with it, and began to stutter at the same time. He was allowed to write with his left hand, and the confusions of orientation and the disturbance of speech rapidly diminished. Fildes and Myers conclude that the child's early visual experience is probably little concerned with the absolute position of seen

9. Sereni, Enrico: Rev. di psicolo **19**:135, 1923.
10. Fildes, L. G., and Myers, C. S.: Brit. J. Psychol. **12**:3, 1921.

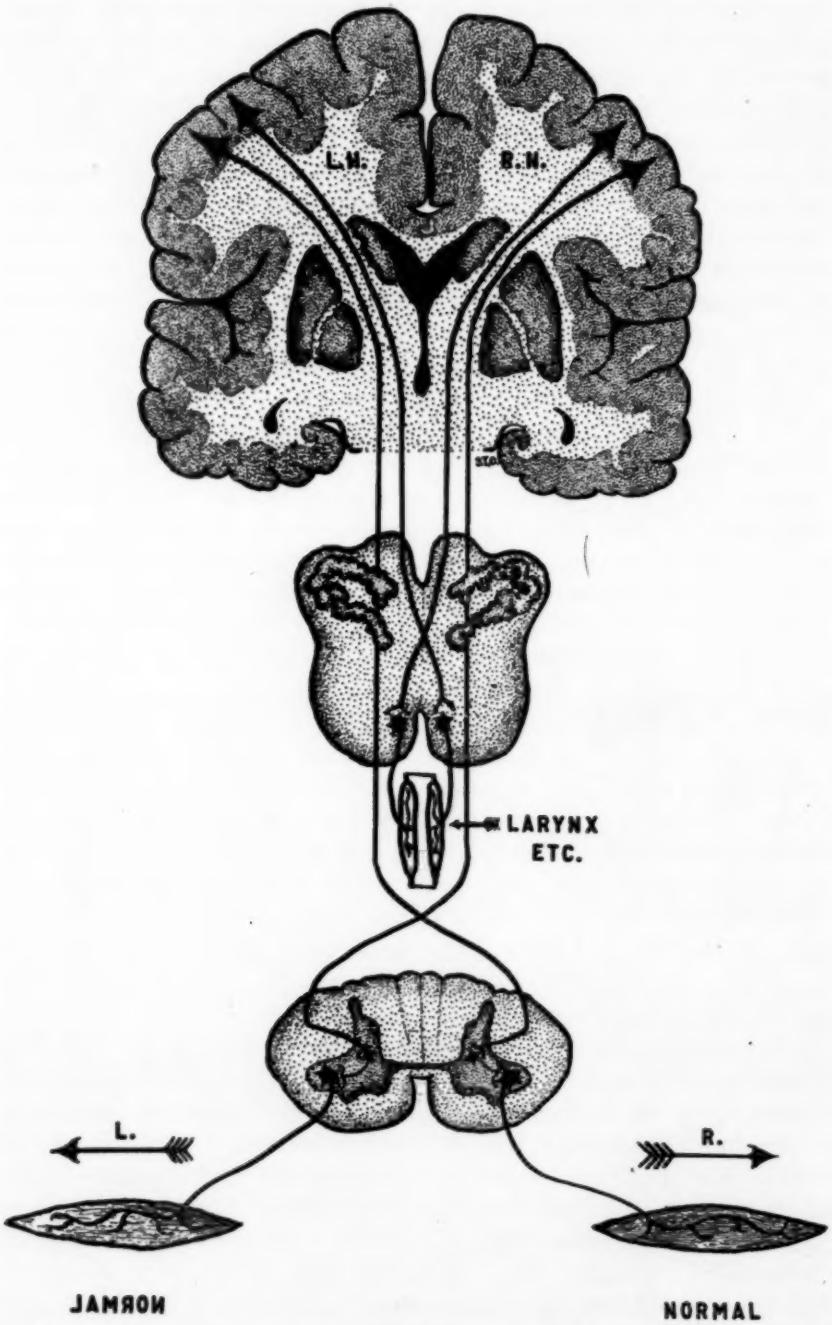


Fig. 9.—Motion of left side exactly opposite that of right in response to innervation of muscles.

objects. His attention is first drawn to form, and his powers of recognition are not gravely disturbed whether that form once learned be represented in the ordinary or reversed or inverted position.

Parson [11] has recently published a book on left-handedness in which he emphasizes the fact that ocular dominance, i.e., the eye used in fixation, corresponds in all except abnormal cases to the "handedness" of the individual. His study is based on the results of experiments with the manuscope, an apparatus—sadly misnamed—to determine which is the dominant or sighting eye. Parson apparently holds that the ocular dominance determines both cerebral dominance and the "handedness" of the individual. His figures are striking in indicating that the facile hand corresponds in side to the eye used in fixation. The selection of one eye as the sighting eye might, however, readily be regarded as a result of the establishment of dominance in one brain hemisphere from other reasons rather than determining it. Parson's manuscope was not available to us until after our observations were complete, and therefore no tests of his method of determining the dominant hemisphere were made on our material.

"CONGENITAL WORD-BLINDNESS"

In the medical literature we find the first record of a case of congenital word-blindness made by Morgan,[12] who reported his observations on a child who could not learn to read, although his vision was normal. He was not mentally defective, and he knew the letters of the alphabet, and Morgan gave to this condition the name of congenital word-blindness because of the similarity in many respects to Kussmaul's [13] acquired word-blindness. Hinshelwood [2] published a monograph on the subject in 1917, in which he reported several other cases and discussed them at length in comparison with the acquired condition. Hinshelwood makes a sharp and, I think, unwarranted distinction between mild and severe grades of difficulty in learning to read. He says: "The term congenital word-blindness ought to be reserved for those grave cases of defect where the difficulty in learning to read was so great and so unusual that it could be regarded without any exaggeration as an abnormal and pathological condition and where the attempts to teach the child to read by the ordinary methods had completely failed." He also says: "The rapidity and ease with which children learn to read by sight vary a great deal. No doubt it is a comparatively common thing to find some who lag considerably behind their fellows, because of their slow-

11. Parson, Beaufort Sims: Lefthandedness, New York, The Macmillan Company, 1924.

12. Morgan, W. Pringle, quoted by Hinshelwood, Footnote 2.

13. Kussmaul: Ziemmsens Encyclopaedia der Speciellen Pathologie and Therapie, Leipzig, F. C. W. Vogel, 1881.

ness and difficulty in acquiring their visual word memories but I regard these slight defects as only physiological variations and not to be regarded as pathological conditions." His envisagement of the etiology of true word-blindness as here described is that of a defective development, in the early stages of embryonic growth, of the special cerebral area subserving visual memory of words and letters.

Our own studies would lead us to believe that while there may be additional factors in certain cases which serve to determine a greater severity of the disability in a given child, these cases as a whole form a graded series, and it is only the occasional child in whom a fair facility in reading is not ultimately achieved and who would therefore fit in Hinshelwood's group. At the present stage of our study, the factor of reversals of individual letters and the tendency to sinistrad reading of letter groups or whole words seems to characterize all of these cases. How many may show a second factor like that exhibited by M. P. in his faulty range of sound associations with the vowels or like the inversions suggested in Jack D.'s case can be determined only by further study. Hinshelwood's conception of a defective development of the cortex destined to become the center of storage for visual word memories is also out of harmony with the more modern conceptions of cortex function. Marie [14] calls attention to the fact that an infant with right hemiplegia never presents aphasia, and he believes that the temporal region, the gyrus angularis and the surrounding zone are not in any sense preformed centers for language, but brain structures adapted by training to that function. Apert,[15] reporting on a case of congenital familial dyslexia, calls attention to the fact that this, like congenital aphasia, is an entirely isolated disability—that is, it is not a part of a general mental defect—and that later the development, which has been checked, recurs. He also has emphasized the fact that children with right hemiplegia are not aphasic, and he expresses the opinion that these developmental delays (hemmungen) are not of anatomic but of functional origin. Pick [16] lists together mutism, infantile agrammatisms, congenital word-blindness and difficulty in the auditory memory as the underlying conditions resulting in check in development of expressive speech in children. Pick also emphasizes that these are not accompanied by a defect in general intelligence, and that after some time normal development proceeds. He states emphatically that these cases are of the group of developmental delays and are not to be considered as defects due to brain lesions.

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14. Marie, Pierre: Presse méd. **17**, March 1, 1922.
15. Apert, E.: Bull. méd., Paris, **38**:9, 1924.
16. Pick, A.: Med. Klin. **20**:21, 1924.

The evidence available from histologic studies demonstrates that there are three distinct types of visual cortical mechanisms, and the data gathered through correlation of clinical studies with the necropsy findings in cases of focal brain disease indicate that there are three corresponding functional levels. Figure 10 shows the distribution of these three different histologic types of cortex on the mesial and lateral aspects of both hemispheres of the brain.

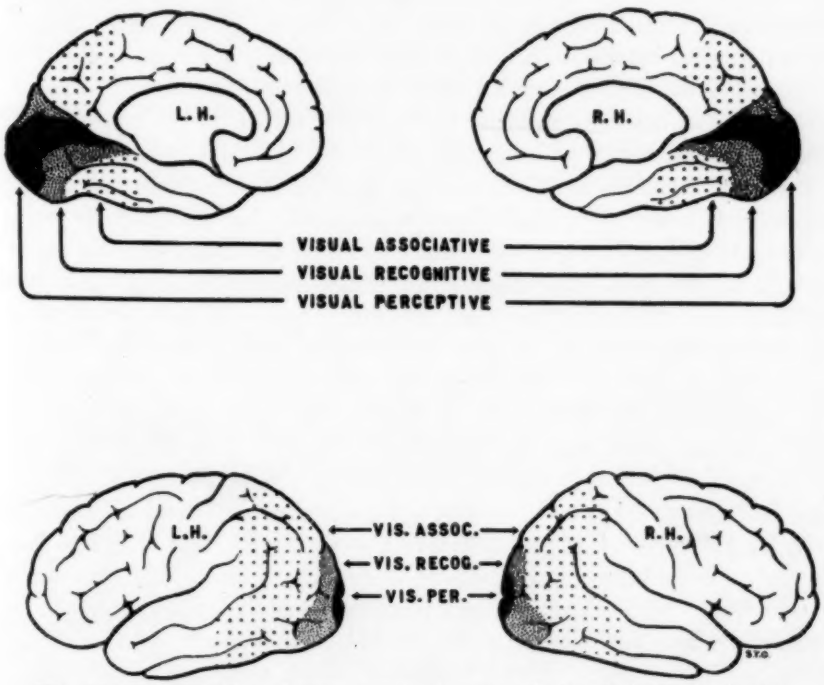

Fig. 10.—Distribution of three types of cortex in hemispheres of brain.

The first of these levels is indicated in the illustration by solid black and by the name "visual perceptive." This is the arrival platform, calcarine cortex or area striata. This last name was given because of the presence of a heavy white band of nerve fibers about midway in the depth of the cortex, which sharply demarcates this field and makes it possible to recognize its exact limits with the naked eye. When bilateral destruction of this cortex occurs, there follows complete blindness. The lower reflex centers are still operative, but no visual impressions of any kind enter consciousness or serve as a control of volitional motor responses. These results do not follow unilateral destruction in this field. This cortex was called the visuosensory by Campbell.[17] The

17. Campbell, A. W.: The Localization of Cerebral Function, Cambridge, University Press, 1905.

term "sensory," however, embracing as it does the whole receptive and recording mechanism, seems too wide for this restricted functional zone, and I have for some time used the term "visual-perceptive" here. This usage will be explained in the discussion of the next level.

The second level is marked in Figure 10 as the visual recognitive and shown by the area of stippling. This is the common occipital type of cortex which practically surrounds the arrival platform. The distribution given conforms to that of Campbell's map and quite closely to Brodmann's [18] area occipitalis, field 18. With extensive losses of this cortex there is retention of mere awareness of visual stimuli but loss of recognition of their meaning. This is the condition known as mind-blindness, which can perhaps best be illustrated by the results of Munk's experiments on dogs, in which it was first recognized and described. After wide ablation of the occipital lobes, Munk found that his dogs were still able to get about without collision with objects—that is, the animal still retained sufficient control of the motor acts by visual stimuli to guide its movements, but it apparently failed to gain the meaning usually carried by such stimuli. Threats with a whip, sight of food when hungry or the sight of its master, except when reinforced by additional sensory data from other fields, evoked no responses. Clinical states of similar type in man have since been recorded in numbers, and from anatomic studies of these, it is obvious that they arise from extensive *bilateral* destruction of the occipital cortices, but only when there is retention of part at least of the visual arrival platform or calcarine cortex. When destruction is unilateral, this complete loss of the capacity to recognize objects does not follow. This separation of awareness of a stimulus from recognition of its meaning is not so completely accepted by the psychologists, who include both of these functions in the term "percept" because of the difficulty of separating them by introspection. The evidence from pathology and histology is so clear-cut, however, that I have come to separate these functions and to restrict the use of the term "perceptive" to that of the first level, i. e., awareness without meaning, and I have so marked the arrival platform on the plate. The second area was called the visuopsychic by Campbell, but the term seems indefensible, and I have used the descriptive term "visual recognitive" for this level.

The third level—the visual associative—is not to be considered a unitary sensory platform in the sense that it is specifically visual in function, as it is in this great posterior association zone that associative interlinking of the data garnered in various sensory fields probably occurs. This field is indicated in Figure 10 by spaced dots, but the area

18. Brodmann: Vergleichende Localizationslehre, Leipzig, Johann Ambrosius Barth, 1909.

so demarcated is not intended to show its exact extent or boundaries, but merely to indicate that those cortices of the association zone which lie nearest the visual recognitive field probably play a preponderantly visual part in the associative function. This area as outlined includes part of Campbell's common temporal and parietal areas, and includes Brodmann's area prae-occipitalis, field 19, area occipito-temporalis, field 37, area angularis, field 39, and part of his area parietalis superior, field 7. There is no such clear mark of identification of the recognitive and associative cortices as we find in the white line of the arrival platform, and microscopic analysis of the architectonic patterns is therefore necessary to determine their extent. Lesions of the third level result in the condition known as word-blindness, in which awareness of objectivity and recognition of the concrete meaning of objects are both retained, but in which the abstract or associative meaning of the printed word is lost. Here, however, for the first time, we see a sharp difference between the results of lesions in the two hemispheres. In both the lower platforms, extensive bilateral lesions are apparently necessary to destroy the respective functions. In the third level, a unilateral lesion is sufficient but only when it occurs in the dominant or lead hemisphere—the left hemisphere in right-handed persons, and vice versa.

To these three separate levels of the visual function, I think that we may tentatively apply the physiologic hypothesis that each related irradiation from one cortical zone to the next must be simultaneous or immediately successive in time, and must be concordant in detail to permit associative linkage. A sensory stimulus which reaches the two arrival platforms results in one conscious impression which we call awareness or the sense of objectivity. We may assume that if the overflow of these impulses into the two recognitive cortices be harmonious in both time and form, the external stimulus will have that correspondence with its respective memory image which constitutes recognition of its meaning. The combined irradiation of the first and second platforms would then result in both awareness of objects and the recognition of their meaning. Activity in the second level aroused by association but without the participation of the first would result in a visual memory, but without the sense of objectivity which characterizes the combined action of both platforms and which serves to differentiate for us the memory image from sensory experience. In the formation of a concept or associative memory, we would postulate the simultaneous or immediately successive irradiation of concordant stimuli into at least three platforms. The arrival platform or visual perceptive would give awareness, the overflow into the recognitive would add concrete meaning of the object or symbol and into the associative would add abstract or symbolic meaning through interlinking with sensory data from other fields.

From the fact that loss of the capacity to read follows a unilateral lesion only when this occurs in the dominant hemisphere may we assume that irradiation is necessary into only one of the two third level cortices to produce a linkage between visually presented symbols and their meaning. That one or the other hemisphere or one locus in one hemisphere must have an initiatory function for all volitional motor responses seems obvious. Were it not for this placing of the lead or control in one side, the two hemispheres might originate opposed or conflicting responses to a given situation. In man's brain the entire initiatory control of certain major functions, such as speech, writing and reading, seems to be in one hemisphere, as is illustrated by the occurrence of the alpha-privitive symptoms—aphasia, agraphia, alexia, etc., following unilateral lesions. Dominance of this degree has not, I believe, been demonstrated in the lower animals, but some form of initiatory control would seem necessary to prevent confusion of responses such as would result if either hemisphere were competent to lead without reference to the activities of the other.

With the recognition of the dominant part played by the associative cortices of the left hemisphere in right-handed persons, attention was drawn from the function of these areas in the right hemisphere. In every study of the subject of volitional speech, writing, etc., the functions of the various areas in the left hemisphere are discussed in detail, as indicated by clinical correlations, but I do not at present recall ever having seen any attempt to envisage the physiologic conditions existing in the corresponding areas of the opposite hemisphere. Many of these cortical fields in the right hemisphere are listed among the "silent areas," and apparently the fact that they are probably activated by incoming stimuli has been entirely overlooked, because attention has been directed to the left hemisphere on account of its dominance and the striking results of interference with this dominance by unilateral lesions. When we consider the visual cortices in the light of their anatomic structure, we must remember that here are structures of almost if not quite equal size, extent and neuron content. In fact, about the only structural difference is that one is the right-left counterpart of the other. I have at my immediate disposal no exact data on the relative weight of the two hemispheres of the human brain, but some years ago, a considerable series of such weights was recorded in the Worcester State Hospital in Massachusetts, and, as I remember these findings, there was rarely a difference of more than 20 to 30 gm. in the size of the two hemispheres, and in several instances this discrepancy was in favor of the right hemisphere from patients who were right-handed, at least so far as writing and the common motor acts were concerned. For many years, as a routine measure, I have cut pieces of cortex from six comparable areas of both the right and left hemispheres, one set as squares and the

other as triangles. These blocks have then been embedded in pairs and cut together so that the slide representing the occipital cortex, for example, in a given case will show a square and triangle, from the right and left hemispheres respectively, mounted side by side. Certain cases, of course, such as paresis and the vascular obliterations, will show marked differences in the two sides, and I have never carried through any accurate studies of the number and development of cells in each hemisphere; but the general impression, gained from the study of several thousand such paired sections, is one of comparative equality. If we follow Kapper's theory of neurobiotaxis, we must consider that the cells of the right hemisphere would not have reached this stage of comparatively full development without constant activity, and that their development is the result of irradiation which takes place equally into all parts of both hemispheres; we must further assume that this irradiation into visual areas of the associative cortex of the right (or non-dominant) hemisphere forms a mnemonic record there.

The exact symmetrical relationship of the two hemispheres would lead us to believe that the groups of cells irradiated by any visual stimulus in the right hemisphere are the exact mirrored counterpart of those in the left. That simultaneous activity in these antitropic nerve cell groups may still give us a single conscious sensation, is shown by Sherrington's [19] study of sensory fusion. He studied visual fusion by means of a special apparatus and came to the conclusion that "during binocular regard of an objective image each monocular mechanism develops independently a sensual image of considerable completeness. The singleness of binocular perception results from union of these elaborated sensations." Cases of amblyopia show us that this fusion occurs only when the images strike retinal areas which correspond through associative training, but when the squint remains a new correspondence is ultimately acquired, suggesting that the fusion of the two images into one conscious impression is a function of education rather than of neuron pattern.

I feel that our evidence as to whether the fusion of the results of stimulation of oppositely oriented nerve cell clusters into a unitary sensation is a function of the arrival platform alone or also obtains in the recognitive zone is not quite precise. However, loss of the ability to recognize the meaning of objects (mind-blindness) occurs only when there are bilateral lesions of the recognitive zone, which suggests that fusion does occur at this level.

The fact that unilateral lesions in the visual associative cortices result in loss of symbolic meaning (word-blindness, etc.) would lead to the conlusion that the mnemonic record contained in the right hemisphere

19. Sherrington, C. S.: The Integrative Action of the Nervous System, New Haven, Yale Univ. Press, 1906.

is not requisite for the formation of a symbolic association, and that at this level fusion between right and left mnemonic records does not occur. The tendency common in young children to mirrored or reversed writing, as reported by Fildes, and as seen in our own cases, and the spontaneous production of a mirrored reversal of letters on first attempts at writing with the *right* hand, as recorded in the case of M. O., all point to the existence in the brain of a mnemonic record in mirrored form which serves as the pattern for these motor expressions. Further, the difficulty in our cases of reading disability in differentiating p from q and b from d and their tendency to confuse palindromic words like not and ton and on and no suggest that the mnemonic record exists in the brain in both orientations.

Letters are in themselves merely objects until they have come to acquire meaning through sound associations or through association in groups of sounds which constitute a word. We would therefore assume that in the process of early visual education, the storage of memory images of letters and words occurs in both hemispheres, and that with the first efforts at learning to read the external visual stimuli irradiate equally into the associative cortices of both hemispheres, and are there recorded in both dextrad and sinistrad orientation. Images of objects require no definite orientation for recognition or differentiation, but when we are dealing with letters which have come by custom to be used in one orientation only, it is clear that the orientation of the recalled image must correspond with that of the presented symbol, or confusion will result.

This suggests the hypothesis that the process of learning to read entails the elision from the focus of attention of the confusing memory images of the nondominant hemisphere which are in reversed form and order, and the selection of those which are correctly oriented and in correct sequence.

Figure 11 gives a graphic outline of this conception. The symbols indicate the fusion into one sensation which occurs in the perceptive cortical platform and probably also in the recognitive platform. As the associative level, the dominant images are given in bold face and the elided ones in outline. The cortical fields are indicated as in Figure 8, except that the arrival platform is marked by the line of Gennari instead of by the solid black.

The frequency, in these cases of reading disability, of reversals of letter pairs, such as we see in M. P.'s gary for gray, of whole syllables, as tar-shin for tar-nish, or of the major parts of words, as tworrom for tomorrow, strongly suggests that there has been an incomplete elision of the memory patterns in the nondominant hemisphere, and that therefore either right or left sequence may be followed in attempting to compare presented stimuli with the memory images, and that this leads

to confusion or to delay in selection. It seems obvious that such confusion would result in a distortion of the motor output in both speech and writing, and if we assume that the additive function of further associative interlinking with auditory and other sensory images must also be simultaneous or immediately successive, we can readily envisage how delay in selection might interfere with the linking of presented visual

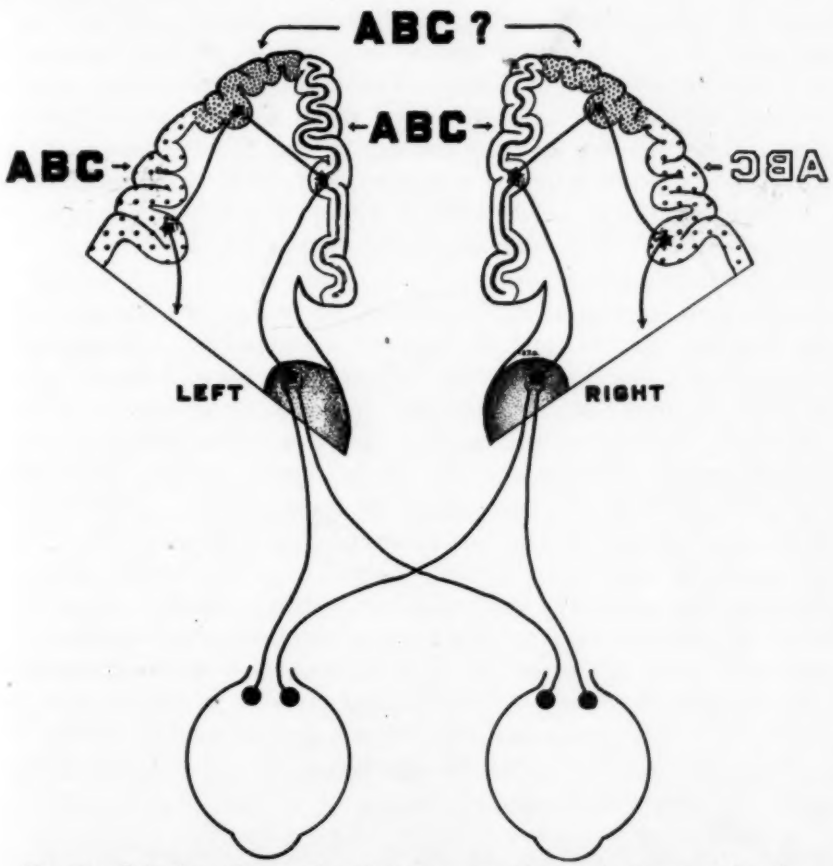

Fig. 11.—Selection of correctly oriented memory images in focus of attention.

symbols with the auditory component of its concept, and hence result in a failure to recognize the meaning, i. e., to read the word as a whole. Most of these children learn to recognize correctly the individual letters, and can also read by name and in proper sequence each letter of those forming a printed word or, as in M. P.'s case, can follow the proper sequence in copying. Here the sequence is determined for them by the external stimulus. When, because of failure in training for automatic elision of the obverse record, however, the mnemonic images may be

recalled in either sequence, they may fail to correspond with the external stimulus, and delay or confusion will follow and may result, as above suggested, in failure of proper association. That training for elision of one set of images may operate in either hemisphere is obvious when we consider that dominantly left-handed children have apparently no greater difficulty in learning to read than do the dominantly right-handed. Those children, however, who are neither dominantly right-handed nor left-handed, or in whom clear dominance has not been well established before they begin to learn to read, probably have more trouble with reversals of letters. The factor of educational method probably also plays a large part here, which will be discussed briefly later. Whether other factors enter into the more severe grades of this disability and hence determine a greater permanence of the condition in certain cases, cannot be determined without much wider observation and experiment. Such enlargement of the study is projected for the immediate future.

The term "congenital word-blindness" because of its association with the acquired condition and the implications therefrom, does not seem to be properly descriptive of this disability, and I would therefore like to offer the term "strephosymbolia" from the Greek words, στρέφω, twist, and σύμβολον, symbol, as a descriptive name for the whole group of children who show unusual difficulty in learning to read. The prefix "strepho" has been chosen to indicate the turning or reversals as it does in the word "bustrophedon," as illustrated above. "Symbolon" is used in its original meaning of "word," "sign" or "token," and not as in Finkelnburg's usage in "asymbolia," in which it included recognition of the meaning of objects as well as of symbols, nor as in the very restricted sense of Oppenheim as a synonym for apraxia. Strephosymbolia thus seems nicely suited to our cases in which our analysis points to confusion, because of reversals, in the memory images of symbols resulting in a failure of association between the visually presented stimulus and its concept.

ERRONEOUS ESTIMATE OF INTELLIGENCE OF DEFECTIVE CHILDREN GIVEN BY PSYCHOMETRIC TESTS

Brief observations on the cases of this reading disability so far studied have brought out several points of psychiatric interest.

It has been pointed out by numerous writers, some of whom have already been quoted in this report, that this difficulty, as is also true of many of the speech and auditory disturbances, is an isolated disability, is not the result of a general mental defect and, further, that often it corrects itself. Nevertheless, there is a strong tendency to characterize these children as "defectives." This has, of course, been furthered by the belief of Hinshelwood and others that there is here a true focal

lack of development in the brain center for visual word memories. Because the term "defective" so constantly implies a general intelligence defect, I have consistently attempted to make use of the word "disability" in describing this difficulty. That the reading disability does not correlate with a low intelligence quotient is obvious from the psychometric ratings of our fifteen cases which show the following distribution:

TABLE 6.—*Psychometric Ratings in Author's Fifteen Cases*

Group	Stanford-Binet Intelligence Quotient	Cases
Superior intelligence	122	1
Average intelligence	91 to 105	9
Dull normal intelligence	85	1
Marginal defective	70 to 75	4

It can be seen by reference to Table 1, that with only one exception all of the children of the lower grades were in the average normal intelligence group or higher but that the four in the higher grades were in the marginal defective or dull normal groups. It might, of course, be inferred from this that only those of inferior equipment failed to overcome their handicap and ultimately to learn to read. I feel, however, from our work with M. P. that this is a distinct problem which will require additional investigation. M. P. had by far the most outstanding case of the series, and I have been far from content, after close personal study, that either the original rating of 71 or the revised rating of 86 really estimated his general intellectual capacity. I think we must therefore challenge the competence of the Stanford-Binet method to give us even an approximate rating in these cases. These children fall in a group of an especial nature more closely comparable to those with true sensory deprivations than to the so-called feebleminded, and there are apparently three factors here which must be considered in judging the adequacy of the test: First, the ratings given are the result of the application of the test to large numbers of children of each chronologic age. In any such group, unless selected on the basis of a reading difficulty, the number of such cases would naturally be small, and we are therefore comparing these handicapped children with an unlike standard. Second, the material of the test itself consists in part of words which are visually presented, and this penalizes their handicap heavily. This factor was an operative one in the change of intelligence quotient in M. P.'s case from 71 on the first examination to 86 on the second. It would seem that a modification of the method might readily be devised to use only auditory presentation except for those parts of the test that deal with images of objects, such as the ball-in-field test, etc., and that this might readily give a better estimate

of their equipment. Third, one path of acquisition of information open to the average child, that of reading, is more or less completely closed to these children. When we realize that M. P.'s disability was so great that practically none of his verbal store had been acquired by vision, we appreciate that his accomplishments in the test are far from establishing as low a capacity as the rating would indicate. This lack of information, however, is not a competent measure of how effectively he can make use of those data which he has garnered by the auditory path.

Several psychiatric reaction patterns were observed in various cases of our series, some of which seem to have a fairly direct derivation from the disability and might readily serve to establish determining character traits.

The first of these was a contented, apathetic disregard of the handicap and its results. M. P. showed this reaction. He was at all times cooperative and entered willingly into all the tests and training experiments, but at no time did he show even a trace of spontaneous interest in his condition or of ambition to overcome his disability. He had apparently made a complete adjustment to his situation and was content to accept himself, as others rated him, as different from other boys; but he had apparently developed no particular feeling of inferiority, nor had he suffered from any emotional blocking because of this difference.

The second type was seen only in outline, and no older cases of similar reaction have yet been encountered. This was a mild paranoid reaction toward the teachers on the part of one or two children who felt that they were being asked to do something that was impossible—as it was for them—when they were expected to keep pace with others in their classes in reading.

The third pattern was brought out with extreme clearness in an adult—M. R., aged 30, whose case is briefly quoted above. She not only had a comparatively severe grade of the disability, but was also of that mild motor incoordinate type in whom many exact motor acts are acquired slowly and with difficulty. She had grown up in a family of brothers and sisters who were probably above average in intelligence and dexterity, so that she had been constantly impressed both at home and in school with the feeling that she was of not quite the same status as the others of her family. The result of this atmosphere was the development in M. R. of an overpowering sense of inferiority which served as an insuperable obstacle to her own efforts. On first examination, she appeared entirely colorless from the emotional standpoint, and was apparently completely submerged by her feeling of inferiority. When, however, her striking reading disability was uncovered, and it was explained to her that this did not necessarily imply a general defect of intelligence, she brightened somewhat, and when she

was asked whether she, herself, felt that she was as incompetent as her family had led her to believe she replied, *"I do not!"* with the first real emotional response elicited.

The fourth type of reaction was observed in varying degree in several children. This was an emotional blocking. M. O. gave the best illustration of this. After she had been in school for some time—making indifferent progress—her mother observed that she was failing in school on work which she knew and made practical use of at home. The mother asked her specific questions on this sort of material on her return from school one day and received the reply, "I don't know," which had become almost a stereotyped reply to all questions on school knowledge. A few moments later M. O. volunteered the exact information for which she had been asked, and when her mother inquired as to why she had not responded before, she replied, "I can tell you, if you will let me tell it myself, but when you ask me I can't tell you anything." This child has since been taught to read with fair readiness for her age, but the blocking of output when anything is demanded as a task or assignment is still very much in evidence in all her efforts.

OVERCOMING OF DISABILITIES BY SPECIAL TRAINING

Little opportunity has as yet been found to review in detail the literature of the teaching of reading, but the hypothesis herein developed concerning these special disabilities would seem to bear heavily on the subject of reading as a whole as well as on these particular cases. Harman [20] accepts the brain defect hypothesis, and calls attention to the fact that practically all of these children learn the ten arabic numerals and most of them learn the twenty-six letters of the alphabet but do not learn to recognize words as a whole, and he feels that this gives the clue to the method for training. He says:

> They must be taught on the plan of the Chinese. . . . A certain mark (Chinese character) conveys to the taught child the idea of a house just as does a picture or as the symbol 1 does the idea of unity or one. To teach these children reading we must fall back on this plan. The word cat must be taken as a whole, not as c-a-t =, but the whole thing is the sign for cat. This method is known as the "look and say" plan, and when carried out by a teacher of intelligence and with great patience, it is possible to teach the child to read. In such cases it is obvious that individual teaching is necessary to secure any effective progress.

Apparently Harman overlooked the logical absurdity in this. Reading whole words at a time is a later acquisition than reading letter by letter, and would seem to be the function of that exact cortical area—the visual memory center for *words*—which Hinshelwood postulates as

20. Harman, U. Bishop: Kelynack's Defective Children, New York, William Wood & Company, 1915.

undeveloped, a theory which Harman apparently accepts. Hinshelwood, in discussing Harman's views, states that he is satisfied that in teaching the word-blind to read the old fashioned methods are preferable to the "look and say" method or, as it is known in this country, the "flash" or "sight reading" method.

The tentative envisagement of the disability herein outlined would suggest that the logical training for these children would be that of extremely thorough repetitive drill on the fundamentals of phonic association with letter forms, both visually presented and reproduced in writing, until the correct associations were built up and the permanent elision of the reversed images and reversals in direction was assured. The flash method would seem from this point of view not only to be inadequate to correct early mistakes in orientation, but also to put these children under an unnecessary and unjust handicap, at least until they had acquired the fundamentals in readily available form. The child has no opportunity to puzzle out whether a symbol means p or q by the flash method, and many such initial errors might well be perpetuated. When a child looks at "not" and reads it "ton," the teacher's first reaction is that the child is inattentive or is not trying, and she is apt to apply either discipline or ridicule, which in turn engender an emotional blocking or a feeling of inferiority without, however, correcting the difficulty. These factors are to some extent illustrated in the case of M. O., which has been quoted above in other connections. She started in school in the first grade at 6 in a room in which the "flash" method of teaching reading was used practically exclusively. She spent two years in this grade, and then advanced to the second only after special coaching in the summer vacation period. In this grade she had a more sympathetic teacher, who gave her more encouragement, and her progress was more rapid although her reading was extremely slow and insecure. In the third grade, she fell into the hands of a teacher who expressed the idea that M.'s trouble was because she was not trying hard enough and attempted to stimulate her progress by pressure. During the first few months of this year, M. O.'s emotional blocking became so great that she was definitely losing much that she had had at volitional command during the preceding year. She was withdrawn from school, and her mother was advised to attempt to train her to read by a return to the old fashioned methods of repetitive drill with painstaking correction of mistakes, and after a few months of this training she had practically corrected the whole difficulty. She still has considerable uncertainty, however, in differentiating on and no, of and for and other short words which were fixed in confused order for her during her period of "flash" training. According to our hypothesis, the training should aim at teaching the child to focus the attention on the correct set of images, and for this purpose the repetitive a-b = ab sort of kinder-

garten drill would seem to offer the most promise. The results of this type of training in M. O.'s case are sufficiently encouraging to warrant the extension of experiments along this line.

It would be of great interest to know whether the comparatively high number of cases encountered in our survey result from any particular characteristics of the teaching method in the local schools, and, further, whether a greater proportion of these disabilities are to be observed in those schools using the flash method than is found in those which still adhere to the older drill methods.

In the table giving the distribution of these cases by grades as found in our survey, it is of interest to note that in the first grade there is only one child—here the teaching is of letters chiefly with only a few simple words. In the second and third, however, in which the reading of more difficult words is added, there is a striking increase in number. From here the cases drop sharply except for the four boys in the seventh, eighth and ninth grades. Our series is of course too small for deductions, but this distribution would suggest that the majority of these children make their own adjustment to this difficulty and learn to read without special training. Some, however, do not, and it would seem as if methods could be devised which will teach those with outstanding cases to read, as well as shorten the period of emotional stress in cases of lesser severity. It is obvious, however, that to be effective such methods must be developed in consonance with a sound neurologic background and be adequately controlled by careful observation and experiments in training. This program we expect to extend in the immediate future.

CLOSED FORAMINA OF LUSCHKA IN THE BRAINS OF THE INSANE

THEIR INFLUENCE ON THE PATHOGENESIS OF THE PSYCHOSES *

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Much work is being done on the circulation, origin and final destination of the cerebrospinal fluid (Weed,[1] Dixon and Halliburton,[2] Dandy and Blackfan,[3] Cushing,[4] Hill,[5] Petitt and Girard [6]), yet little or no attention has been given to the types of obstruction or partial obstruction existing in the course of the fluid through the ventricular outlets. Special consideration is here given to the foramina in view of the fact that some authorities consider the ependymal foramina of the fourth ventricle as normally closed and as playing no important rôle in the circulation of the cerebrospinal fluid. If correct, such views would mean that there are two entirely separate fluid areas; one in the ventricular systems and one in the subarachnoidal spaces, completely separated except for the tissue spaces by means of which the ventricular fluid seeps through the brain substance. Since such views (von Monakow [7]) have been used to

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* Read at the Fortieth Session of the American Association of Anatomists, Buffalo, April, 1924.

1. Weed, L. H.: Studies on the Cerebrospinal Fluid, J. M. Res. **31**:21 (Sept.) 1914; The Production of Meningitis by Release of Cerebrospinal Fluid, J. A. M. A. **72**:190 (Jan.) 1919; The Development of the Cerebrospinal Spaces in Pig and in Man, Contributions to Embryology, No. 14, Carnegie Institution Publications **4** and **5**:224-225, 1916-1917.

2. Dixon, W. E., and Halliburton, W. D.: The Action of the Choroid Plexus on the Secretion of the Cerebrospinal Fluid, J. Physiol. **40**:30 (March) 1910.

3. Dandy, W. E., and Blackfan, K. D.: An Experimental and Clinical Study of Internal Hydrocephalus, J. A. M. A. **61**:2216 (Dec.) 1913.

4. Cushing, Harvey: Studies of the Cerebrospinal Fluid, J. M. Res. **31**: 1 (Sept.) 1914.

5. Hill, Leonard: Physiology and Pathology of the Cerebral Circulation, London, Churchill, 1896.

6. Petitt, A., and Girard, J.: Sur la fonction sécretoire et la morphologie des plexus choroid des ventricules lateraux du systéme nerveux central, J. M. Res. **31**:95 (Sept.) 1914.

7. Von Monakow, C.: Eine neue Form von Dysgenesie der Plexus Choroidei laterales als morpholog. Basis der dementia praecox (katatonie), Libro en Honor de D.S. Ramon Y. Cajal **2**:159, 1922.

develop special theories of the physical basis of psychoses, it becomes important to show in a decisive way the exact conditions of the foramina in the brains of the insane.

The initial purpose of this investigation was to determine in adult human material the closed or open condition of the lateral apertures of the fourth ventricle (foramina of Luschka). It was soon found that pouchlike closures of these foramina existed in many of the brains of the insane as well as of some normal persons. Such closures would obstruct one of the main avenues of flow of the cerebrospinal fluid from the ventricular system into the subarachnoid spaces. The further purpose of this investigation was to determine whether any pathologic processes in the brain could be correlated with long standing closures, and to what extent such conditions might be interpreted as due to insufficient circulation of the cerebrospinal fluid over the cerebral surfaces.

The literature dealing with the subject is scanty. Thus far, a gleaning reveals the fact that only eight cases of closure have been reported: Luschka, one case; Retzius, three cases; Hess, two cases; Virchow, one case; and Sutton,[8] one case. Retzius[9] examined 100 brains and Hess examined fifty-four; the other authors do not report the number of cases examined. Some of the foregoing authors interpret the closures as probably of embryonic origin; others believe that they may be of pathologic origin. No one hitherto has attempted to collect any extensive material with a view to determine whether any abnormal structural changes could be associated with closed foramina.

It soon became evident that the material might yield significant information in this direction. Therefore each specimen with a closed foramen was examined with special reference to the conditions of the choroid plexuses, ventricles, ependymal lining, foramina of Luschka and Magendie, subarachnoidal cisternae, sulci, gyri, meninges, cerebral vessels, arachnoidal granulations, brain substance, basal nuclei and thalamus.

MATERIAL

The material on which this investigation was made consisted of 901 brains; 666 were from insane persons, 150 from epileptic and eighty-five from presumably normal persons. Nearly all these brains were well preserved in solution of formaldehyd. Twelve brains were examined at necropsy before they were subjected to fixatives. Eighty-seven closures of the foramina of Luschka were found; sixty-seven of these were unilateral and ten bilateral. In brains of the insane the percentage

8. Sutton, Sir John Bland: Choroid Plexuses and the Ventricles of the Brain as a Secretory Organ, Lancet **1**:1143 (June) 1923; The Lateral Recesses of the Fourth Ventricle, Their Relation to Certain Cysts and Tumors of the Cerebellum, and to Occipital Meningocele, Brain **9**:352, 1887.

9. Retzius, G.: Das Menschengehirn, Stockholm **1**, 1876.

of closures was 11.6; in brains of epileptic persons, 5.3; in normal brains, 2.5. The percentage for the normal brains is no doubt high, for this so-called normal material came from the dissecting room, and, as shown by Todd,[10] this is for the most part made up of socially ineffective persons with a low grade of mentality.

METHODS

The gross method used in determining the closed foramina was that of inflation. A small glass tube was placed in the fourth ventricle, either by elevating the cerebellum and inflating through the foramen of Magendie, or in the medium cut specimens, the tube was placed directly in the lateral extension of the ventricle from the median side. Either procedure gave the desired results, for when the foramina were closed they were easily inflated. To be further assured of the fact of closure, the brains were submerged in water and then inflated. Such a procedure reveals, by the escape of a bubble of air, the slightest rupture of the lateral recess. Just sufficient pressure to inflate was ordinarily used, but some of the larger pouches were subjected to a great pressure, with no signs whatever of air escaping (Figs. 1, 7, 8, 9, 11). A few of the closed recesses showed a small leak when inflated and submerged. These brains (nine in number) were examined with a binocular microscope, and the small openings were found to be mechanical ruptures, usually in the form of a split or straight tear. These mechanical ruptures, in every case, were found on the floor or ventral wall of the pouch, usually in the center, at which place the normal opening does not exist. Here also the possibilities for mechanical injury in removing the brains from the cranial cavity and later during examination are very great. The borders of these pouches were firmly attached, uninterruptedly throughout, and in not a single instance were the plexuses found visible, either by gross examination or with the aid of the binocular microscope. Such results clearly show these as abnormalities, for in the normal lateral recess the plexus at all times projects freely from the sickle-shaped opening on the upper anterior edge of the lateral extensions. To be further assured that the pouches were completely closed, a number of them were removed with surrounding structure, cerebellum and medulla, and these were sectioned 10 microns thick. The sections show the extension of the lip from the medulla at the region of its greatest breadth (Fig. 6). The lip is made up of a plate of nervous matter to which the pouch is attached throughout uninterruptedly. In the same figure the plexus can be seen hanging freely from the roof of the pouch.

10. Todd, T. Wingate: Cranial Capacity in Social Ineffectives, Anat. Rec. **27**:222 (April) 1924.

Another procedure used in determining such closures was that of injections of paraffin into fresh brains. Figure 4 shows a specimen into which hot paraffin colored with Congo red was injected three hours after death. The injection was through the foramen of Magendie and under considerable pressure. The pouchlike extensions of the lateral recesses were distended and showed no evidence of rupture. Both recesses were closed, as can be seen in the illustration.

DEVELOPMENT AND HISTORY

These closures, cystic in appearance, are unperforated expansions of the posterior medullary velum extending laterally from the rhomboidal fossa.

The early formation of the lateral recesses is well known. The roof of the embryonic fourth ventricle at first is a complete ependymal covering, nowhere perforated. About the middle of the second month when the pons flexure is completed there is a flexure of the roof as well as of the rhombic lip on each side forming the early recesses. According to Blake,[11] the lateral recesses become perforated in man about the end of the fourth month or later and establish communication between the ventricular system and subarachnoid spaces.

From histologic appearances of the choroid plexuses it cannot be denied that some fluid is formed within the ventricles in early embryonic life. At that time the ventricles are relatively large, the brain wall is thin, cell proliferation is ependymal and the great migrations of cells to form the cortex are still under way. That the ventricular fluid may pass directly through this thin brain wall and play a part in the growth processes as conceived by von Monakow is plausible. This mode of egress is limited as the brain wall thickens. That there is an active secretion by the plexuses after the fourth month, is indicated by the pouchlike bulging of the recesses, as if from some internal pressure, which leads to their rupture.

After perforation the lateral recesses lose their distended character and the roof and floor in many cases come in contact, except where they are separated by the protruding choroid plexus. It is further evident that from this time on the lateral recesses recede from the cranial wall and are confined to an area just dorsal to the roots of the glossopharyngeal and vagus nerves and ventral to the flocculus and its peduncle, and their openings appear as slits, as can be seen in a child at term.

In the adult the floor of the recess is formed chiefly from the secondary lip where it extends from the medulla at the region of its greatest

11. Blake, Joseph A.: The Roof and Lateral Recesses of the Fourth Ventricle, Considered Morphologically and Embryologically, J. Comp. Neurol. **10**:79, 1900.

width (Fig. 6). Here it forms a plate of non-nervous matter, which extends to the extremity of the recess and is convoluted and folded on itself as it enters into the formation of the roof and choroid plexus (Blake). In the normal development, the lip at its extremity does not turn over into the roof but ends as a free rounded margin. In the adult it appears as a typical sickle-shaped opening through which projects the voluminous end of the plexus (Figs. 2 and 5).

In the lower mammalia there is usually a slight extension of the epithelium of the roof beyond the choroid plexus forming a lateral wall for the recess. Cannieu evidently observed this arrangement, and concluded that the recess is closed in all mammals. It seems that his observations are correct for the caudal part of the recess but not for the cephalic part, since here there is a complete disappearance of the wall of the recess, and thus a free communication with the subarachnoid cavity.

The lateral recesses and their connection with the subarachnoid spaces were considered first by Key and Retzius.[12] Their monograph is prefaced by an historical review, in which due credit is given to preceding anatomists, Bockdaleck, Reichert and Luschka. Their real nature was first detected by Key and Retzius (1875). The patency of the foramina is described by them as a normal condition. In no instance did they find a membrane stretched over the end of the plexus and attached to the sides, thus closing the recess by a blister-like or bubble-like projection. They later (1896) found such a condition in three brains out of 100 examined, and described such closures as being due to a continuation of the wall itself by stretching. Hess[13] examined fifty-four lateral recesses and found but two closures. It should also be noted that Hess describes the brain of a child at term, in which he states that the ventricle was closed by a pouchlike fold of the pia. He also describes a thinning and perforation of the sides of this pouch.

Luschka demonstrated the patency of the foramina by injection of lime masses. He at first described one closure which he thought was a pathologic prolongation of the vascular membrane that covers the upper end of the flocculus. A similar closure was later described by Retzius, which was not pathologic but a continuation of the thin wall itself. He seemed to think these closures are embryonic and not pathologic.

Virchow considered the pouch an example of a partial cystlike widening of the fourth ventricle (hydrocele ventriculi), or, more probably, an embryologic closure with a secondary bubble-like widening. Sutton

12. Key and Retzius: Studien in der Anatomie des Nervensystems und des Bindesgewebes, Stockholm, 1875, first half.

13. Hess, C.: Das Foramen von Magendie und die Oeffnungen an der Recesses laterales des vierten Ventrikels, Morphol. Jahrb. **10**:578, 1885.

described a similar specimen in which the pouch was the size of a cherry and in which it arose from the lateral recess. The presence of the foramina of Luschka is admitted by almost all workers in the field, and also it is admitted that they serve as normal outlets for the ventricular fluid.

The existence of the foramen of Magendie has been debated and even denied by many authors, in particular Cannieu, Cruveilhier and Kölliker. Three cases of its absence in men have been described by Cannieu and Gentes. When the foramen of Magendi does exist as a normal condition, it is quite variable in size. In many cases the foramen is filled completely by the caudal protrusion of the choroid plexus of the fourth ventricle. Several times during my observations in the region of the obex, it was necessary to dissect away a portion of the surrounding structures to be assured of its patency. In order to be satisfied as to the normal relation of this foramen to the medulla and cerebellum, brains were examined at necropsy before being placed in liquor formaldehydi or other fixatives. I have found that the foramina of Magendie are usually present as perforations of the posterior medullary velum, but in the adult human brain the channel by means of which they communicate with the cisterna magna is considerably restricted by the tonsillar lobes. The medulla and the upper part of the spinal cord are closely pressed against the cerebellum, thus adding resistance to the passage of the cerebrospinal fluid through the foramen of Magendie. In some cases when the lateral apertures were small the foramen of Magendie was large. The size of the foramina of the fourth ventricle is variable, and we might expect such compensatory changes to exist in them. The lateral apertures are usually large and distinct, while the median aperture is restricted.

In 901 adult brains of normal, insane and epileptic persons, I have found the following to be the normal condition of the lateral apertures. The lateral recesses of the fourth ventricle, one on either side, lie in the shallow depression formed between the flocculus and the biventer lobule of the cerebellum, at the level of the junction of the pons and medulla. The roof of the recess lies partly under the posterior border of the flocculus, and partly under the anterior inferior border of the lobule biventer. The floor of the recess rests on the glossopharyngeal and vagus nerve roots, and in a few cases slightly cephalad to them, but not as high as the auditory nerve. The anterior and posterior borders are formed mostly from the floor with its corresponding attachments to the flocculus in front and the biventer lobule caudally. The size of the lateral apertures varies only a little in the brains with the typical open foramina. The remains of the rhomboidal lip, which forms the floor of the recess, normally ends in the sickle-shaped opening through which projects the plexus anteriorly.

With regard to the dilated and imperforated lateral recesses the position in general is the same, with the following difference: The larger the pouchlike recess, the more it comes to lie in a position more cephalic and directly under the flocculus. In the partly distended but open recesses, the foramina are usually found on the anterior lateral surface, with the plexus protruding anteriorly. These findings are in line with those of Blake on the human brain. The distended lateral recesses vary greatly in size and shape. In those brains in which the foramina were absent, the recesses were in many cases as large as 20 mm. wide by 25 mm. long (Fig. 3); by length I refer to its lateral extension. Some were as small as 6 mm. in width by 9 mm. in length (Fig. 7); others, being long and narrow, 5 mm. wide by 18 mm. in length (Figs. 8 and 9). A great number were round; some were large, 18 mm. by 18 mm. (Fig. 1); others smaller, 14 mm. by 15 mm. (Fig. 11), which seems to be the average size of the distended recesses.

In a number of cases of closures, the roofs of the distended lateral recesses were dissected from their cerebellar attachments, and in no instances were they found patulous. The patency or open condition of the distended lateral recess is a normal anatomic and physiologic condition. When the recesses are closed, it is due to an overdevelopment of the secondary rhomboidal lip, with failure to perforate during the fetal period.

NATURE AND FUNCTIONS OF THE FLUID

Many explanations have been given regarding the nature and function of the cerebrospinal fluid, yet nothing definite is known of the exact rôle it plays in nerve cell metabolism. The cerebrospinal fluid is present in all vertebrate brains, and its normal physiologic activity, especially its circulation, is essential to the vital processes of the mammalian brain. It is a fluid which makes possible the physical laws of osmosis within and around the nervous tissue, and the rapid removal of waste products. In composition it resembles Locke's modification of Ringer's solution (Halliburton [14]). It possesses a trace of sugar and a small percentage of protein. The sugar present is greater in amount in the ventricular fluid, while the protein content of the cerebrospinal fluid is greater in the extraventricular fluid and is increased in many diseases (Boyd [15]). Although the fluid is simple in character, great cytologic changes are encountered in diseases of the nervous system.

Several explanations of the nature and function of the fluid have been proposed. One is that it acts as a water cushion to the brain,

14. Halliburton, W. D.: The Possible Functions of the Cerebrospinal Fluid, Brain **39**:213 (Oct.) 1916.

15. Boyd, William: Physiology and Pathology of the Cerebrospinal Fluid, New York, The Macmillan Company, 1920.

serving as a protection to the central nervous system. This is in all probability partly true; yet when we consider the position of the larger subarachnoidal spaces and cisterns, we find that those parts of the brain which are more subjected to blows or external contusions possess the smallest subarachnoid spaces (temporal, parietal and occipital regions). The position of the larger cisterns is due to the general conformation of the cranial contents. Therefore this view is wholly inadequate.

Another view is that of Mott,[16] who regards the cerebrospinal fluid as the lymph of the brain. However, in composition it is different from ordinary lymph. Lymph contains an amount of protein almost equal to that of the blood, while the cerebrospinal fluid contains only a trace of protein. Anatomists today have agreed that there are no lymph vessels in the central nervous system, but the perivascular spaces are regarded as suggestive of this lymphatic function. The lymphatic system serves as a middle man between the blood and the tissues, conveying to the tissues the oxygen and nutritive substances they need, and returning to the blood stream the waste products of cell activity, which are eliminated by the organs of excretion. We do not know the amount of fluid entering the subarachnoid spaces from the perivascular channels. We do not know the exact difference in the composition of the fluid formed from the choroidal plexuses and that from the perineural and perivascular channels. A procedure for such a determination would no doubt be difficult. Yet we do know that the extraventricular fluid (fluid found in the subarachnoid spaces) contains more albuminous compounds and a higher percentage of carbon dioxid. This is referable to active physiologic metabolic processes within the nerve cells, the end products of which reach the subarachnoid spaces by way of the perineural and perivascular channels. The very composition of the extraventricular fluid itself is suggestive of one which acts as the true physiologic fluid. This fluid constantly bathes the cells of the central nervous system and conveys from them to the blood stream their waste products as a result of cellular activity. In this way we may look on the cerebrospinal fluid as the lymph of the brain. Yet the true lymph derived from the extensive capillary beds in the nervous system is found in the spaces around the nerve cells. This pericellular fluid comes to the surface through the perivascular spaces from which the waste products are poured into the general fluid system, and thus carried to the pacchionian granulations.

If one considers the central nervous system as the most highly specialized system in the body, it follows that a very efficient system

16. Mott, F. W.: The Oliver-Sharpy Lectures on the Cerebrospinal Fluid, Lancet, July, 1910.

is necessary for the removal of its waste products. The fluid that is continually being secreted by the choroid plexuses into the lateral, third and fourth ventricles must be regarded as the source of the bulk of the ventricular fluid and as a means of maintaining pressure and active flow through the ventricles, apertures, subarachnoid spaces and pacchionian granulations.

CIRCULATION OF CEREBROSPINAL FLUID

Various estimates have been made as to the amount of fluid, which normally seems to be from 90 to 130 c.c., varying somewhat with the individual. This fluid is being secreted continually and is escaping continually, so that the total daily volume exchange is considerable. Increase in the amount of fluid is due to oversecretion of the choroid plexuses or to some interference with the absorptive mechanism. That part of the fluid with its origin from the plexuses in the lateral ventricles passes by way of the two interventricular foramina of Monro into the third ventricle, where it receives accretions from the small plexuses within this ventricle. From the third ventricle it passes through the sylvian aqueduct into the fourth ventricle, again receiving accretions from the choroid plexuses of the fourth ventricle. From the fourth ventricle it escapes to the subarachnoid spaces by way of the two lateral apertures (the foramina of Luschka) and the median aperture (the foramen of Magendie).

The percentage of fluid passing from the fourth ventricle into the subarachnoid cisterns by way of the median aperture and the two lateral apertures is not definitely known. The early experiments of Luschka showed that injections of viscous fluids almost to the point of hardening can come through the lateral recesses from the inside, irrespective of the fact that the foramen of Magendie is open. This shows how much more readily the fluid passes through the lateral apertures than through the median aperture. Such results can be verified easily, for macroscopic examination of the foramina will show the lateral apertures much larger than the median aperture. This is especially so of the brains at early necropsies, when the brain is as near its normal condition as one could expect.

A comparison of the lateral apertures with the median aperture as to development, growth and position of the surrounding structures may lend some clearness to this question. Blake found that the choroid plexus of the fourth ventricle develops first in the lateral apertures, and in a normal person they project freely from the lateral apertures into the subarachnoid space. The median plexus develops after the lateral plexuses have formed and joined with each other at the midline. If the secreting cells of the choroid plexuses are considered as a functional unit of the cerebrospinal fluid system, it is evident that the lateral apertures

play a greater rôle in the circulation than does the median aperture. This is especially so in the human brain in which the cerebellum is greatly expanded backward and compressed over the dorsal surface of the medulla. The tonsillar lobes and uvula tend to compress the median foramen so as to almost obliterate it or at least greatly impede the flow through any channel that may exist in that area. This restriction does not occur in the region of the lateral apertures. Here the great growth of the brachium pontis and the lateral lobes of the cerebellum with a rudimentary condition of the floccular lobe on the one hand tends to increase the size and patency of the lateral foramina; on the other hand, the great growth of the semilunar and tonsillar lobes of the cerebellum in the fifth month of embryonic life tends to close over and restrict the foramen of Magendie. This may be one of the factors which conduces to the rupture of the lateral recesses.

With regard to position, the lateral apertures are more in the direct course of the circulating fluid than is the median aperture. The sickle-shaped openings of the lateral recesses project anteriorly, and are in a direct line with the circulating fluid. The fluid from the median aperture passes into the cerebellomedullary cisterna and then over and around the cerebellum. A small part of the fluid probably passes down the cord dorsad as far as the lumbar cisterna and returns by way of the ventral surfaces of the cord to the basal cisterna, where it again intermingles with the bulk of the fluid as it courses through these cisternae around the medulla and pons. The bulk of the ventricular fluid passes through the free communication of the lateral apertures with the basal, pontile and cerebellar subarachnoid cisternae. The subarachnoid channel around the pons and in the notch is shallow. It seems evident that the large basilar artery in the midline on the ventral side of the pons, and the cerebellum, great cerebral vein and pineal body on the dorsal side would serve as a complete or partial separation of the fluid from the two sides. From these large subarachnoidal spaces beneath the tentorium cerebelli the greater part of the fluid passes upward through the tentorial incisura either to the right or to the left, and under the cerebral hemispheres, thence over the surfaces of the sulci and gyri of the cerebral cortex to reach the pacchionian granulations. As it bathes the brain, it receives through the numerous perivascular channels the pericellular lymph derived from the capillary beds in the brain substance.

The cerebrospinal fluid is continously removed by filtering through the pacchionian bodies into the venous sinuses of the dura mater, as was shown first by Key and Retzius, and later by many other workers in the field (Hill, Weed). It was held by Key and Retzius that the escape takes place by way of the pacchionian bodies, which are simple sacculations of the subarachnoid tissue protruding into the dural venous sinuses. This has been demonstrated by Weed in his several important con-

EXPLANATION OF PLATE

Fig. 1.—Brain B 1420 (B indicates Binghamton material). Ventral surface of brain showing lateral recess of the fourth ventricle; *a*, closed pouchlike extension of lateral recess; *c*, cerebrum; *cr*, cerebellum; *p*, pons.

Fig. 2.—Brain B 1455. Typical sickle-shaped extension of the rhomboidal lip, forming the ventral and lateral borders of the lateral recess; *n*, normal sickle-shaped opening; *x*, choroid plexus projecting anteriorly and laterally from the sickle-shaped opening. Glossopharyngeal and vagus nerves have been pulled forward to expose plexus.

Fig. 3.—Brain B 1954, showing *a*, closed pouch on right side; *n*, normal sickle-shaped opening on left side; *ar*, arachnoid membrane in normal position covering pouchlike extension of the recess. The thick extension of the rhomboidal lip is much more dense and firm than the arachnoid membrane. The position of the glossopharyngeal and vagus nerves is normal, always immediately under the pouch and quite adherent to it; *b. art.*, basilar artery.

Fig. 4.—Brain B 2448. Both lateral recesses closed. A hot red paraffin mass was injected through the foramen of Magendie. Fresh brain at necropsy; *a*, closed recess.

Fig. 5.—Brain C 673 (C indicates Cornell material). Showing normal projection of choroid plexuses from the lateral recesses; the ninth and tenth nerves have been pulled back in order to expose the plexuses.

Fig. 6.—Brain B 1472. Section of closed lateral recess showing extension of rhomboidal lip attached uninterruptedly throughout; *rh*, rhomboidal lip extending from the medulla at the region of its greatest width and forming the pouchlike closure; *m*, medulla; *cr*, cerebellum; *x*, choroid plexus hanging from the roof of the closed pouchlike extension of the lateral recess.

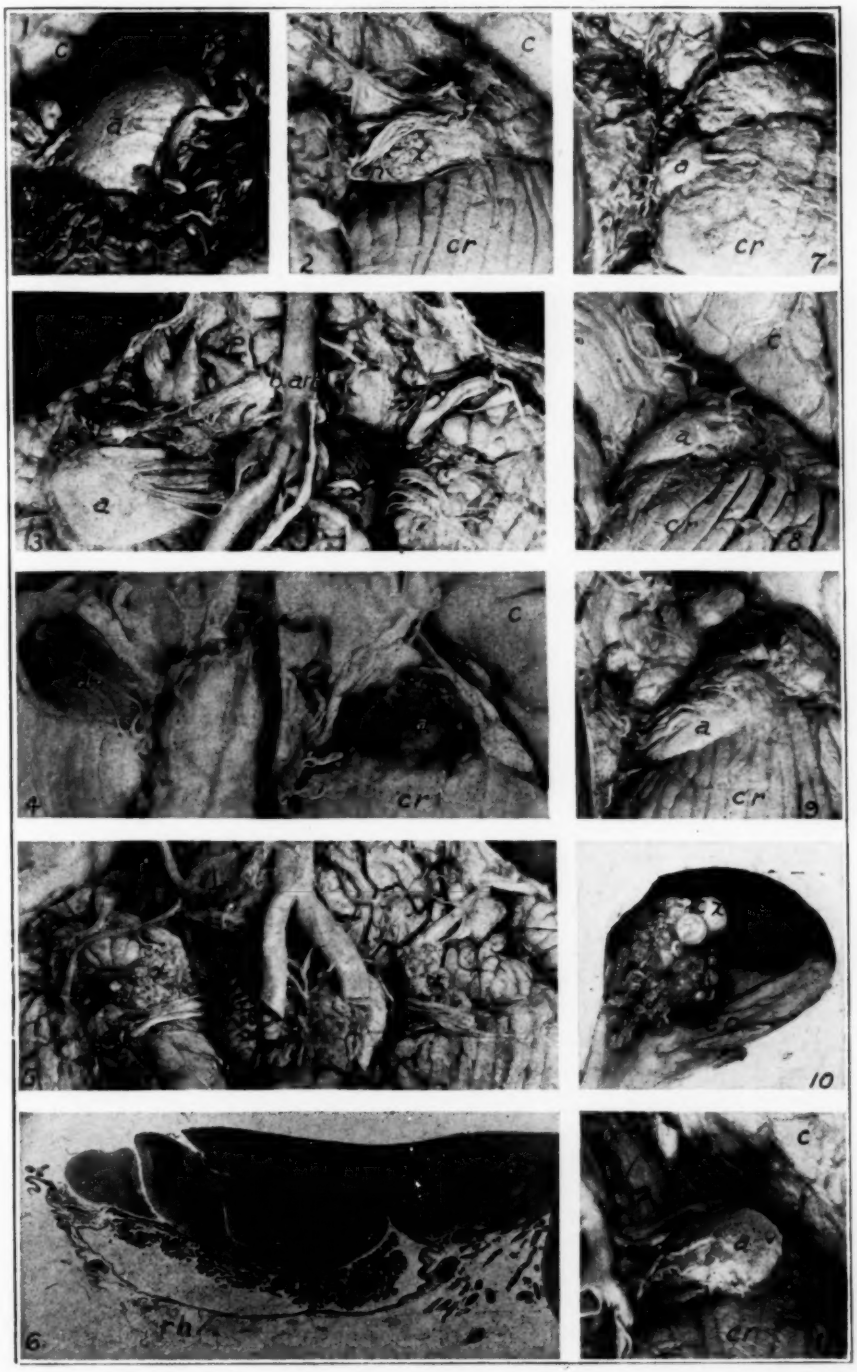
Fig. 7.—Brain B 1360. Small pouchlike closure. The arachnoid has been dissected from the pouch and surrounding area.

Fig. 8.—Brain B 1602. Another type of closure, long and narrow.

Fig. 9.—Brain B 1472, showing position of ninth and tenth nerves, immediately under the pouch and always attached to the outer membranous part of the pouch, also showing relation of arachnoid as in Figure 3.

Fig. 10.—Brain B 1954. Photograph of posterior horn of lateral ventricle; *cx*, cystic plexus, note the absence of villi over the cystic areas; *ep*, wrinkled ependymal lining of the ventricle.

Fig. 11.—Brain B 1425. Average size closed pouch.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tributions on the subject. A certain amount of the fluid is being secreted continually by the choroid plexuses; thus, we have new accretions continually to the extraventricular fluid. By such additions to the fluid, the waste products do not remain long in the circulation, but are carried along by the bulk of the fluid to their place of absorption into the blood stream. If the amount of circulating cerebrospinal fluid were smaller, no doubt the waste products would be much longer on their course to the point of entrance into the blood. It is easily conceivable that a total or partial absence of the circulating fluid in any region would permit the accumulation of waste products in that region, resulting in marked deterioration of the corresponding brain substance. This appears to be the case when obstruction, even when only partial to the normal circulation, exists. The amount of cellular deterioration depends on the nature and degree of the obstruction, or on the lack of circulation to which the degenerated areas were subjected.

VENTRICLES

The ventricular system was studied in every case of closed foramina, and dilated or partly dilated ventricles were a common occurrence in the brains examined. The lateral ventricles were found to be noticeably dilated in 70 per cent. of the cases of closed foramina. The size of the ventricles varied from the slightest distention to that of a ventricle five times the normal size. The ventricles with slight distention were not recorded, and the 70 per cent. included only those which were noticeably distended. In sixteen instances of brains with complete closures of the foramen of Luschka on one side, I found the lateral ventricle distended far more on the side of the closed foramen. In thirteen of these sixteen cases the closure was on the left side, and the left lateral ventricle was found to be much larger than the right. In four cases of unilateral closure, I found the ventricles dilated more on the side opposite the closure. In these four instances the closures were all on the right side, and the ventricles were much more distended on the left.

It seems from such results that the left lateral ventricle is more subjected to distention than is the right. At any rate we may conclude that a unilateral closure of the foramina of Luschka on the left side is in almost every instance accompanied by a distention of the lateral ventricle on that same side, while a closure on the right side is accompanied either by distention on the left side or by equally distended ventricles of both sides. The ventricles were found to be normal in size in seventeen cases of closed foramina, the larger percentage of these unilateral closures being on the right side. It seems from such results that a left-sided closure affects a left-sided distention more than a right-sided closure affects distention on the right side. In ten brains the closures were found on both sides (Fig. 4). In these ten bilateral

closures the left lateral ventricles were found to be larger than the right in two instances. In five cases the ventricles were equally distended on both sides. In the three remaining cases of bilaterally closed foramina, the ventricles were found to be normal in size.

The distended ventricles as a whole may be due primarily to obstruction within the corresponding ventricular outlet or within the absorptive mechanism; they may be a result of hypertrophy of the choroid plexuses or of an oversecretion of the cerebrospinal fluid; or the distention may be the result of a combination of such conditions. In the cases which have been studied the distention of the ventricles seemed to be secondary, a result of atrophy of the corresponding brain substance. With a circulation of fluid over the cortex which is insufficient to remove the waste products that accumulate there by way of the perivascular channels, we may expect stagnation of fluid. With this stagnation we find marked atrophy of the brain substance. The cranial content is a fixed quantity. Once we have a loss of material in any one region, we can only expect a compensatory increase in the bulk of the surrounding cranial contents. The ventricular system is one wholly suggestive of such behavior. It is, therefore, evident that in a vast majority of such cases the ventricular dilatation is secondary and compensatory to atrophy of the brain substance as a result of improper circulation.

EPENDYMA

The most striking feature of the ependymal lining of the ventricles is that the pitting or wrinkling follows exactly the distention of the ventricles (Fig. 10). When the ventricles are enormously dilated, the ependyma is greatly pitted and wrinkled, and when the ventricles are normal, the ependyma is normal or only slightly pitted. In cases in which the lateral ventricle on one side was the larger, the ependyma was correspondingly more pitted and wrinkled on the same side. In cases in which the distention was more pronounced in the anterior horns, the ependyma in that region was noticeably more pitted. The same is true of the third and fourth ventricles. In no instance did I find distention without wrinkling of the ependyma, and in not a single case did I find a ventricle normal in size with the ependyma pitted. The conclusions drawn from such observations are: 1. In the normal condition of the ventricles, the ependymal surface is smooth and follows closely the general contour of the surrounding brain substance. 2. In the distended ventricles, the ependyma is subjected to considerable stretching, corresponding to the increase in the size of the ventricles. 3. In these cases of pitted ependyma the most plausible explanation seems to be that the walls collapsed after the pressure had been removed, the walls returning in part to the original position in which they were before they were subjected to stretching.

CHOROID PLEXUSES

Cystic degeneration of the choroid plexuses in the lateral ventricles is common, being found in a large percentage of cases as described by Findley.[17] So frequently are cysts present that Faivre in 1855 described "choroid vesicles" as normal and peculiar to the human subject. Findley examined sixty-five brains of the insane, and found cystic plexuses present in 50 per cent., while Ogston (cited by Findley) found them in only 19 per cent. of the brains of known drunkards. In the seventy-seven brains with closed foramina which I have examined, the plexuses were cystic in fifty-four; twenty-eight were markedly cystic on both sides, while thirteen presented larger and more prominent cysts on the same side as the closed foramina. In nine of these cases the plexuses were slightly cystic, usually at the extreme end of the plexuses in the posterior cornu of the lateral ventricles. In three brains with double closures of the foramina of Luschka the plexuses were cystic on one side. In one instance the cysts were larger on the side opposite the closed foramen.

These cysts are racemose and vary in size from that of a pinhead to the size of a cherry seed. In several cases I have found larger cysts, the size of a cherry. Luschka describes them as being as large as a hazelnut, but Findley did not encounter any quite so large. The walls of these so-called cysts were very thin, with a smooth shining surface, like those described by Findley. The small bladder-like cysts number from eight to as many as thirty in the plexuses of the posterior horn of the lateral ventricle (Fig. 10). These numerous capillary convolutions of the vascular pia vary in size. The small bladder-like cysts are usually present in the form of a bunch of grapes. In many cases we find one or two large cysts with several smaller ones. These large cysts of the choroid plexus were found especially in brains with distended ventricles. In more than 100 brains with dilated ventricles the plexuses were cystic in all but six. Monakow has correlated pathologic conditions of the plexuses with various kinds of psychoses. From my gross observations, it is evident that the cystic plexuses are associated with and are almost always present in brains with dilated ventricles, particularly those in which the dilatation is a result of marked atrophy of the brain substance.

PACCHIONIAN GRANULATIONS

Observations were made on the pacchionian bodies in an attempt to determine their position and number, and to determine whether they played any important rôle in those cases of unilateral closures of the

17. Findley, John W.: The Choroid Plexus of the Lateral Ventricles of the Brain, Their Histology, Normal and Pathological (in Relation Specially to Insanity), Brain **22**:161, 1899.

foramina of Luschka. The pacchionian granulations were examined in sixty-five brains having closed foramina of Luschka. In seventeen cases with unilateral closures of the foramina of Luschka, the pacchionian granulations were more numerous, and in many instances larger, on the side opposite the one with the closed foramen. This we may interpret as being due to a greater amount of fluid on the side with the open foramen. In thirty-four of the brains the determination was doubtful, since the granulations seemed to be equally distributed on both sides. In eight bilateral closures, the pacchionian granulations were equally distributed on both sides. In two instances of double closures, they were more numerous and more prominent on the left side. In only four cases were the granulations more numerous on the same side on which the closure existed.

Apparently some correlation can be made between this "absorptive" mechanism and the closed foramina. For example, in a right-sided closure it seems evident that the right side would not receive as much fluid as the left. What is still more probable, on the side in which we find the greater amount of fluid, we would expect a more efficient mechanism for the absorption of the fluid.

BLOOD VESSELS

Examination of the blood vessels was also made in the brains with the closed foramina. Of the sixty-five brains examined for conditions of the vessels, forty-eight were found to be atheromatous, one half of these being extremely atheromatous and sclerotic. These aneurysmal dilatations were found especially prominent in the basilar and vertebral arteries, and in the circle of Willis. In seventeen instances they were found to be normal as far as any pathologic condition could be detected macroscopically.

The cystlike conditions of the choroid plexuses are associated with the atheromatous condition of the blood vessels. In forty of the brains with atheromatous vessels the plexuses were markedly cystic, while in only four cases of atheroma the plexuses appeared normal. In seven instances the blood vessels were normal and the plexuses were normal. In six cases the blood vessels were normal and the plexuses cystic.

MENINGES

A marked thickening and cloudy appearance of the pia-arachnoid were found in twenty-eight of the brains with closed foramina. Of these twenty-eight cases of probable leptomeningitis, eighteen were found more pronounced in the frontal regions. The pia-arachnoid here was extremely thick, cloudy and of a dull milky appearance. The greatest thickening was found in the anterior part of the frontal region, gradually becoming thinner and losing the cloudy appearance in the parietal

regions. In only a few cases was the arachnoid cloudy or thickened over the occipital region. The pia-arachnoid was much thicker and more cloudy on the same side of the brain as the closure in six of the probable cases of leptomeningitis. The thickening of the arachnoidal membrane was the same in all regions in three cases. In only one instance did I find the pia-arachnoid thicker and more cloudy on the side opposite the closure (Brain 1302). In this brain the pacchionian granulations were sparse on the side of the leptomeningitis. This seems to be an explanation of its thickening and cloudy appearance, as it probably caused a stagnation of the fluid.

It can be safely assumed that the greater amount of fluid is absorbed on the side in which the pacchionian granulations are more numerous. It can be considered that normally the perivascular channels are evenly distributed on both sides of the brain, and that the same amount of waste products is emptying into the cerebral subarachnoid cisterns over both hemispheres. Then with a circulation of the fluid insufficient to carry away these waste products on one side, we might expect changes in the surrounding tissue as a result of stagnation of the fluid and an accumulation of the toxic products given off from the nerve cells as a result of their activity. This pronounced thickening of the arachnoid on the side of deficient pacchionian granulations is perhaps the results of such a condition. It is especially noticeable in the frontal regions where the circulation is probably less active. The thickening is also more pronounced and there is a greater deposit of foreign material in the region of the deeper sulci, or we may say the cortical subarachnoidal cisterns. In all these brains the most noticeable changes were in the arachnoidal membrane over the sulci rather than the gyri, perhaps because of a larger amount of stagnant fluid enclosed within the sulci and naturally a greater accumulation of toxic material.

In some of these cases of chronic leptomeningitis the pia-arachnoidal thickening is probably the result of past inflammatory conditions, and consists mostly of hyperplastic connective tissue. In other cases it is produced not so much by active inflammation as by continued and oft repeated disturbances of circulation and nutrition, and in particular by fluid stasis. Ziegler [18] states that the disease is most frequently encountered in the frontal regions on the anterior part of the cerebrum. It is less pronounced in the posterior regions (occipital), yet he gives no explanation for this.

Weed, by the intravenous injection of a sufficient number of virulent organisms within the meninges, was able to produce typical lepto-

18. Ziegler, Ernst: Special Pathological Anatomy, trans. from the eighth German edition by Donald MacAlister and H. W. Cattell, New York, The Macmillan Company, 1896-1897.

meningitis if the cerebrospinal fluid was withdrawn during the artificial septicemia. This tends to show that the leptomeningitis is due in part to a lack of cerebrospinal fluid. Thus it is evident that leptomeningitis may be the result of improper circulation of the cerebrospinal fluid. The waste products resulting from cellular activity within the brain substance are being continually emptied into the subarachnoid cisterns, and unless these waste products are properly removed many pathologic conditions may exist within the surrounding tissues.

GENERAL PATHOLOGIC CONDITIONS

In the brains with the closed foramina the striking characteristic was that the atrophies, lesions, softenings and general loss of brain substance in 95 per cent. of the cases were on the same side of the brain as the closed foramina. In thirty-five of the brains the atrophy of the brain substance in general, as shown by sulci and gyri, basal nuclei, etc., was especially pronounced on the same side as the closure. In thirteen cases the pronounced loss of brain substance was the same in both hemispheres. In eight brains atrophy of the frontal regions was pronounced, and the same on both sides. These cases of general atrophy are not included in the thirty-five cases of atrophy on one side. In four cases, lacunar softenings were found on the same side as the closure. In one brain lacunar softenings were found on the opposite side. The closure was on the left side; the lacunar softenings were on the right side, yet very small. The brain, however, showed other pathologic conditions on the left side, such as dilated lateral ventricle, cystic plexus and atheromatous vessels. In this brain the right side appeared to have been closed; the right cerebellum was damaged, yet a tough pouchlike covering, or extension of the embryonic rhomboidal lip, was present, into which I was unable to gain entrance with a trocar for the purpose of inflation.

In one brain with bilateral closures of the foramina of Luschka, lacunar softenings were found in the right lentiform nucleus; a large softening was found in the right inferior parietal region extending deep into the brain substance as far as the posterior cornu of the lateral ventricle, involving the tail of the caudate nucleus and the radiations of Gratiolet. In nine brains with bilateral closures of the foramina of Luschka, both the right and left hemispheres showed about the same pathologic conditions. The atrophies, lesions, softenings, etc., were pronounced in every case in both hemispheres. Other conditions, such as pontile pigmentations, found in fifteen cases, and discoloration of the brain substance associated with degeneration were also characteristic of many of the pathologic conditions.

In three cases the substance seemed to be normal on the side of the closed foramen. In brain No. 1426, the closure was on the right side.

The ventricles, plexus, gyri, sulci, blood vessels and brain substance all appeared normal. The only condition suggestive of improper circulation of the cerebrospinal fluid was that the pia-arachnoid was much thicker in the frontal regions. In brain No. 1850 the closure was on the left side. The basal nuclei on the right side were entirely destroyed by hemorrhage, which seemed to be extraventricular, involving the lateral ventricles only by secondary displacement, by pushing the roof and all of the ventricle down and inward. There were no noticeable disturbances of the brain substance on the left side; however, the plexus was quite cystic, the ependymal lining of the lateral ventricle was greatly pitted and the vessels were extremely sclerotic. In brain No. 1702 the closure was on the left side. This was a peculiar closure since the surrounding arachnoid inflated with the lateral aperture of the ventricle, yet it showed no evidence of patency when inflated and submerged in water. The brain substance was apparently normal. The most striking condition was the very small atheromatous blood vessels, which no doubt greatly impeded the necessary circulation through the brain. The vertebral arteries were almost closed; the lumen was extremely small.

In seeking an explanation other than extraneural toxemias for such pathologic conditions, one of two factors presents itself for consideration. There may be either an insufficient nutrition of the brain cells from a deficient blood supply, as in arteriosclerosis, or there may be an improper removal of waste products accumulated from cell activity, as in plexus sclerosis or in cases of closed foramina. When any area of the brain is cut off from an adequate blood supply, it gradually dies. The same conditions may come about from an accumulation of waste products. The dead tissue is destroyed chiefly by the phagocyte cells of the brain itself, and is removed by way of the cerebrospinal fluid. By obstruction of the foramina or of the pacchionian granulations, or by any other condition which interferes with the normal flow of the fluid, the waste products are allowed to accumulate, and a vicious condition is established. With such progressive degenerative changes we may expect gross impairment of the cerebral tissue and a loss of mental faculties.

SUMMARY AND CONCLUSIONS

The observations recorded in the foregoing paragraphs offer evidence that in the normal person there exists free communication between the ventricular system and the subarachnoid spaces. This means of communication is through the foramina of the fourth ventricle, complete absence of which results in hydrocephalus. Absence of the foramen on one side may result in marked degenerative processes in the corresponding cerebral hemisphere.

According to von Monakow, there is no direct communication between the ventricular system and the subarachnoid spaces, except by

large glial ependymal spaces. If I interpret correctly the often complicated statements of this author, he practically denies the existence of the foramina of the fourth ventricle as functional structures in the adult, since he found them closed in the fetuses examined. This author is of the opinion that the fluid elaborated by the choroid plexus of the ventricle passes directly through the brain wall in all directions by way of the small glial ependymal spaces, and in cases of diseased plexuses introduces toxic or improperly detoxified substances into the brain tissues, which are the causes of psychoses.

The observations recorded in this work do not wholly accord with this view so far as they prove that the foramina in the lateral recesses of the fourth ventricle do exist as functional structures, even in the majority of the insane. In most cases the foramina are plainly evident and demonstrable as the partial extension of the ependymal rhombic lip, which ends as a free rounded margin through which projects the choroid plexus. Such normal foramina as outlets for the ventricular fluid (Fig. 2) are definite in outline, and in no way can they be mistaken for mechanical ruptures of the membrane. Such a normal communication can be seen even before the removal of the arachnoid membrane, at which time the normal condition of the foramina is undisturbed. Therefore the idea that the ventricular fluid normally seeps through the substance of the brain wall to gain the subarachnoid must be denied, at least for the bulk of the fluid, in most adult brains, even in the insane.

When the foramina were closed, as was found in eighty-seven cases studied (seventy-seven brains), such closures were imperforated lateral expansions of the posterior medullary velum, forming a complete pouch which in no way communicated with the subarachnoid spaces. Such closures are regarded as pial ependymal diverticula which failed to perforate during late embryonic life. We may then regard the patency of the lateral recesses of the fourth ventricle as a normal embryologic and physiologic condition, and when closed as an overextension of the rhomboidal lip which has failed to perforate. In this sense a closed foramen is an abnormal condition. Evidently such closures resulted in partial obstruction to the normal ventricular outlets of the cerebrospinal fluid, since unilateral abnormal conditions of the brain substance could be correlated with the closed foramen of Luschka on the same side.

The ventricles of the brain were noticeably distended in fifty-five cases of closed foramina. In sixteen of these cases, the distention was greater on the same side as the closure. This may be interpreted as being secondary to cerebral atrophy resulting from insufficient circulation of the cerebrospinal fluid and a failure to carry away the waste products which accumulate in the subarachnoid spaces.

The choroid plexuses and the ependymal lining of the ventricles showed marked hyperplastic changes in fifty-four of the seventy-seven

brains with closed foramina. In thirteen instances such conditions were more pronounced on the same side as the closure. We may regard the choroid plexuses as the secretory units of the cerebrospinal fluid circulatory system, serving also as a protective membrane in that they do not permit the passage of harmful products into the cerebrospinal fluid system. According to the view of von Monakow, the breaking down of the choroid plexuses allows any toxins within the blood stream to pass through, thus causing a toxemia which results in degenerative processes of the brain substance. He says the severe plexus conditions are in proportion to the severe degenerative conditions of the brain substance, and attributes to them a biochemical position of primary importance in the development, the integrity and the normal action of the nervous system. Kitabayashi [19] believes that in cases of schizophrenia, lesions of the plexuses may precede and at least partially determine those of the cerebral cortex.

There can be little doubt that the choroid plexus secretes a large part of the cerebrospinal fluid. However, it must be stated that Hassin [20] is of the opinion that the plexus is an organ for absorption of the fluid, a view contrary to that of von Monakow and recent experimental evidence. But it seems evident that his histopathologic findings do not wholly bear out or substantiate his interpretations of the plexus as being absorptive rather than secretory in function. The histopathologic changes which he found in the subarachnoid in cases of meningeal carcinoma and meningitis, as he admits, wholly support the fact that the flow of the fluid is from the brain substance to the external surface; if not, the subarachnoid spaces would not be infiltrated with pathologic cells while the parenchyma of the brain remained normal. Experimental evidence does not show the course of any substantial amount of the fluid from the brain substance through the ependymal wall into the ventricles. Results showing such a course of the fluid have been obtained under conditions of high pressure and then such a passage of the fluid was scant (Wislocki and Putnam [21]). Hassin further says that it would be hard to understand such pathologic conditions if the choroid plexus were an organ of secretion of the cerebrospinal fluid. This we cannot agree with, since the histopathologic conditions studied in hyperplastic changes of the connective tissue indicate its secretory function and are in no way indicative of an absorptive process. The inside of the cystic

19. Kitabayashi, Sidanichi: The Choroid Plexuses in Organic Diseases of the Brain and in Schizophrenia (a Critical Review by Minkowski), J. Nerv. & Ment. Dis. **56**:21, 1922.

20. Hassin, G. B.: Notes on the Nature and Origin of the Cerebrospinal Fluid, J. Nerv. & Ment. Dis. **59**:113 (Feb.) 1924.

21. Wislocki, G. B., and Putnam, T. J.: Absorption from the Ventricles in Experimentally Produced Internal Hydrocephalus, Am. J. Anat. **29**:313, 1921.

formations or hyperplastic plexuses shows a considerable amount of granular material, which is itself suggestive of a true, cystlike formation rather than absorption. If it were an absorptive process there would be at least as much granular material on the outside as on the inside. Aside from experimental evidence and physiologic necessity, there are innumerable points in the histologic structure of the choroid plexuses, embryonic, normal and abnormal adult plexuses that indicate its secretory nature. Likewise the hyperplasia of the plexuses may be due, at least in part, to the large number of *Corpora amylacea* found around the blood vessels. Such histopathologic conditions are found within the plexuses, and are indicative of changes within the plexuses, rather than outside of them.

The pacchionian granulations, through which the cerebrospinal fluid is absorbed into the blood stream, were found in seventeen cases of unilateral closures to be larger and more numerous on the side opposite from that on which the closure existed. This is interpreted as evidence that more fluid is circulating on the side of the open foramen, and thus a more extensive absorptive mechanism (granulations) would necessarily be developed.

The pia-arachnoid was greatly thickened in twenty-eight of the brains with the closed foramina. This thickening (and in many cases a decided appearance of noninfectious chronic leptomeningitis) was more pronounced in the frontal regions. The thickening or hyperplastic condition of the arachnoid was from the pial side, and is interpreted as being due to a lack of cerebrospinal fluid circulating over the frontal region, and thus being a stagnation of fluid with accumulated waste products which induced the hyperplastic condition in the membranes.

In brains with closed foramina the most striking features, taken as a whole, were atrophies, lesions and general loss of brain substance. In 96 per cent. of these cases these were on the same side as the closure. A few such conditions were bilateral, but the majority, and those which showed the greatest deterioration of brain substance, were on the same side as the closed foramen.

The findings in the choroid plexuses, in the ventricles, in the ventricular foramina, in the cerebral cortex (especially in the cases of closed foramina) and in the pacchionian granulations all tend to confirm the view that the part of the cerebrospinal fluid originating from the choroid plexuses has three definite and distinct functions: (1) the maintenance of a constant pressure; (2) the addition of bulk or quantity to the fluid; (3) the function of washing the brain surface and carrying away the waste products which result from functional activity of the brain cells. Under normal as well as abnormal conditions, these waste products for the most part originate in the brain substance, and by means of the perineural and perivascular spaces reach their ultimate destination in the

subarachnoid spaces overlying the cerebral cortex. If such products are allowed to accumulate, even in small amounts, they become toxic and impose a noxious effect on the surrounding parenchyma.

In addition, there are undoubtedly toxemias of vascular origin, as described by von Monakow, who attributes these toxic conditions to the hypogenesis, sclerosis and parenchymatous degenerations of the choroid plexuses. On the other hand, we may conceive that in certain cases such toxemias may arise *sui generis*, due to insufficient bathing of the nervous tissues and a failure to carry away their metabolic waste products. Such an explanation may be applied at least to cases of failure of the choroid plexuses to develop, described by von Monakow, and to cases with closed foramina as described in this article. It seems that in such cases the extensive reservoir of fluid into which waste products from the brain substance are poured by the perivascular spaces has a circulation not sufficiently active to maintain normal conditions.

A diminished supply of the fluid may be due to hypogenesis of the plexuses (von Monakow), disease or sclerosis of the plexus, occlusion of the foramina of Luschka and Magendie, obstructive conditions in the meninges or insufficient or defective absorptive surfaces in the arachnoidal granulations. In the cases with diseased plexuses, toxemias of vascular origin as conceived by von Monakow cannot be denied. When, however, unilateral closure of a foramen of Luschka occurs, the corresponding unilateral degenerations of cerebral substance call for further explanations. Such cases strongly suggest that the bulk of the cerebrospinal fluid maintains a bilateral distribution over the two hemispheres of the brain. Along the medulla and in the tentorial notch the basilar artery on the ventral side, and pineal body, great cerebral veins (of Galen) and the falx on the dorsal side tend to maintain this bilateral system. In such cases the diminished supply of fluid, with an increased toxemia due to a greater concentration of waste products, offers itself as a likely explanation of unilateral degenerations corresponding to a closed foramen.

The evidence here presented points in one direction, namely, that the marked unilateral deterioration of nervous substance observed in these cases may be interpreted, at least in part, as the result of inadequate circulation with stagnation of the fluid, and an accumulation of waste products due to such a type of obstruction as a closed foramen of Luschka on the same side.

IS ENCEPHALITIS AN INFECTIOUS, EPIDEMIC DISEASE?

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Since the recent epidemic of influenza, lethargic encephalitis has appeared in this country and abroad in what is considered an epidemic form. Sporadic cases are frequently seen. Much experimental work on the cause of this disease has been done, but up to the present time, practically nothing has been established. It is significant, I believe, that the pathologic changes are chiefly about and within the small blood vessels and capillaries. Congestion and engorgement occur first, and cloudy swelling of the nerve cells appears only secondarily; later, cytolysis may occur. It is evident that the virus, whatever it be, enters the brain by way of the blood stream. This is not in harmony with the usual conception of intracranial infections, excluding the granulomas.

While encephalitis is thought to be an acute, infectious, epidemic disease, certain facts argue strongly against this: First, the unusually long prodromal stage. While an acute onset is often reported, careful inquiry into the history of patients who recover usually reveals a gradual onset, sometimes extending over weeks or months. Few patients will be found to have been in good health prior to the onset of the disease. One of my patients, Case 1, showed character changes ten months before taking to bed. Second, the prolonged and irregular course of the disease. All definitely proved epidemic diseases are self-limited. They terminate spontaneously at a definite and known time. Lethargic encephalitis has no known limit. No two cases are alike; the disease may last from a few days to weeks or months. Third, the varied pathology and the presence of successive pathologic stages observed at necropsy. All stages, from early capillary engorgement to sclerosis, are found. This suggests a continuation of the virus and is at variance with our accepted picture of acute infectious diseases. Fourth, no cases of suppuration have been reported. All infectious and epidemic diseases are associated or complicated in a certain percentage of cases by suppuration somewhere in the body. This suppuration, it is true, is often due to a secondary invader; nevertheless, this is the natural history of all epidemic diseases due to a bacterial cause. The exception to this statement is poliomyelitis, the cause of which is still in doubt; it may yet prove to be of the same general nature as encephalitis.

If lethargic encephalitis is not due to bacterial infection, what other causes could be operative? A toxemia with specific selective action

would well explain the clinical picture. It would be in line with Rosenow's theory of the adaptation of bacteria and their toxins according to changes in their environment, and also with Vaughn's theory that the varied symptomatology of febrile diseases is due to the selective action of the protein toxins liberated by bacteria. Pribam [1] of Prague called attention to the fact that the influenza of 1918 affected largely the gastro-intestinal tract, and that this epidemic was followed by a large number of cases of encephalitis. He conceives of encephalitis as an allergy. Regardless of the explanation accepted, the point from which the protein toxin or protein sensitizing substance enters the circulation is, in my opinion, the biliary tract.

Since 1922, when my attention was first directed to the subject, I have observed a series of cases of encephalitis with distinct infection in the gallbladder. These patients were treated for the most part surgically, with remarkable improvement in the encephalitis and clinical recovery in a few days. This experience has been sufficiently definite to justify the assumption that the encephalitis present in these cases was due to selective action of a toxin caused by infection within the gallbladder. Certainly, the experience is sufficiently definite to lead to study by others.

REPORT OF CASES

CASE 1.—*Clinical History.*—Miss E. G., a nurse, aged 26, seen first on Dec. 4, 1922, complained of pain in the upper part of the abdomen, occurring usually two hours after meals; this was relieved sometimes, but not always, by food. The condition had existed for eight months. The patient frequently vomited to relieve distress; recently, vomiting had been more marked, the pain more constant and closer to meals. The stools were clay-colored; no tarry stools were noted. The weight was stationary at 165 pounds (74.8 kg.). The patient had been taking a powder which gave relief for a time only. She had had typhoid fever at the age of 7; measles and diphtheria in childhood; the appendix had been removed five years before admission, but the operation had not given complete relief from symptoms.

Examination.—The general examination revealed nothing of importance except a slight enlargement and tenderness of the liver, two fingerbreadths below the costal arch. Laboratory tests showed grayish-black stools, which contained no blood. The stomach contents contained no free hydrochloric acid; the total acidity was 23; no blood, sarcinae or Oppler Boas bacilli were found. The urine showed no abnormalities. Gastro-intestinal roentgenograms showed no changes except a spastic colon.

Diagnosis.—A diagnosis of gallbladder disease was made, and was supported by the report of the surgeon who had performed the appendix operation five years previously which showed that the gallbladder was adherent to the stomach; it was opened and closed after finding no stones present. A note was also made that enlarged glands were present along the cystic and common ducts.

1. Personal communication from one of his students.

Neurologic Findings.—The patient entered St. Vincent's Hospital on December 20. Shortly afterward it was noted that she was depressed; memory was poor, and she was drowsy and irritable. Shooting pains in the abdomen and legs were complained of; jerking motions of the arms, legs and fingers were noted, and she complained of jerking of the abdominal muscles. The abdominal reflex on the right disappeared, and the left knee jerk could not be obtained. On December 25, she complained of diplopia and distortion of objects, and the white uniform of the nurse appeared black. On one occasion, she saw only the upper half of my body as I entered the room. Delirium appeared at midnight, and for two or three nights in succession she fought with her nurse to come to my office to keep an appointment which she declared had been made. Spinal puncture revealed the fluid to be under a slight increase of pressure. Examination showed no globulin; the Wassermann and gold chlorid tests were negative; the cell count was 11.

Course.—The condition improved for two or three days after spinal drainage and then became worse, when another spinal drainage was made. Vomiting now became persistent and severe, and always contained bile. The upper right rectus became rigid, and tenderness increased. The white cell blood count was 8,200.

Because of the definite gallbladder picture and the persistent vomiting, operation was performed, Jan. 2, 1923, by Dr. Fred Douglass. A markedly thickened gallbladder, buried in a mass of adhesions, was found and removed. No other pathologic changes were noticed. Recovery from the encephalitis was prompt and complete.

On questioning classmates of this nurse, it was found that changes in her character, disposition and mentality had been noticed by them as far back as February, 1922.

Case 2.—*Clinical History.*—Mrs. I. C., a widow, aged 32, had an attack of sharp pains in the left chest with slight fever in June, 1923. The condition was diagnosed as pleurisy. She left the hospital after two weeks not completely relieved. She then noticed that she was getting weak, easily became fatigued, was forgetful and did not do her work satisfactorily. When I saw her, about July 1, she complained of attacks of severe shooting pains in all parts of the body, jerking of the arms and legs and distorted vision, objects appearing long and narrow. The body temperature was 100.

Examination.—Examination showed slight nystagmus to the right, and moderate bilateral ptosis. The teeth had all been extracted; the tonsils were moderately red and inflamed; the heart was normal; the lungs showed no signs of pleurisy; the abdomen was markedly tender in the upper right quadrant with rigidity of the upper right rectus muscle, and Murphy's sign was positive. There was definite jerking of the abdominal muscles, the deep reflexes were slightly exaggerated, and the abdominal reflex was not obtained.

Course.—A diagnosis of encephalitis was made. The pain in the upper right side of the abdomen became more marked, and extended to the shoulder. Food caused distress, and the stools became clay-colored. Nonsurgical biliary drainage was therefore undertaken. The *B* bile was intensely black and showed a considerable amount of pus, mucus and red cells; it contained also a motile organism resembling *Lamblia intestinalis.* Eight or ten of these could be seen in each field.

The patient was then sent to the hospital, and the distended gallbladder was removed by Dr. Fred Douglass on July 13. The appendix also showed signs of inflammation, and was removed. Twenty-four hours after the operation, the patient remarked that for the first time in months things seemed natural to her.

Previously, everything had been unreal; things had been distorted, and she had been annoyed constantly by dreams in her wakeful moments.

CASE 3.—*Clinical History.*—Mr. B. F. L., a merchant, aged 41, had been under my care since 1915, when he had had repeated atatcks of tonsillitis and also a moderately severe psoriasis which had existed for more than ten years. The tonsils were removed in April, 1915, by Dr. A. L. Steinfeld. Within a few days the psoriasis began to disappear, and within a week was gone. In February, 1920, this man reported again because the psoriasis had reappeared. He had also severe neuralgia, extending out from the region of the sixth dorsal vertebra along the left to the front. He complained of nausea, marked epigastric distress and constipation. The stools were clay-colored, and the tongue was coated. Nonsurgical biliary drainage was undertaken. The *B* bile was green and thick, contained much mucus but very little pus; many cocci were seen. Both the psoriasis and the neuritis disappeared after a course of five drainages.

The present illness began early in July, 1923. The patient noticed slight pain and discomfort in the left buttock, extending down the thigh and leg. About August 18, he came to the office for relief. The pain was definitely along the course of the sciatic nerve, and did not involve the hip joint. The Achilles reflex was absent, and there was marked tenderness around the head of the fibula. The patient complained also of a sense as if there were a lump in the epigastrium and of mild nausea on arising in the morning. He had lost about 10 pounds (4.5 kg.) in weight during the preceding six weeks.

Examination.—General examination revealed nose, throat and teeth in excellent condition; the heart and lungs were normal. The abdomen showed marked tenderness over the ninth costal border; the entire right rectus muscle was rigid; Murphy's percussion over the costal border produced epigastric pain. The abdominal examination was negative otherwise.

Course.—In the absence of any other source of infection and with the knowledge of past biliary tract involvement, the present gastric distress again seemed to point to the biliary tract as the source of infection. Nonsurgical drainage was again attempted, and on four successive occasions only a little *A* bile and a very small quantity of *C* bile was obtained; no *B* bile was secured at any time. No improvement was noted.

The patient was then lost sight of for about three weeks, but was seen again on October 9, when it was learned that he had become progressively worse. He had lost considerable weight, and the skin was definitely subicteric. On October 10 a marked change was noted. The man was drowsy, but could be awakened readily. There was a definite masklike expression; the man was emotional; euphoria was present, and diplopia had been present all day. The mother stated that he had used rather strong language during the day, something which he had never previously done in her presence. The tongue was coated, and marked tremor was noted. The pupils showed nothing unusual. The liver was now palpable about two fingerbreadths below the costal arch and was slightly tender. The abdominal reflexes on the left side were absent, on the right diminished; the right knee jerk was exaggerated, the left diminished.

On October 11, the drowsiness had increased, and bilateral ptosis was present. Operation was performed by Dr. Fred Douglass, and a greatly enlarged, distended and what appeared to be a constricted gallbladder was removed. On opening the gallbladder, more than 2 ounces (59.2 c.c.) of thick, viscid, mucilaginous green bile was removed; and six or seven, apparently freshly formed stones, each about the size of the head of a small pin, were found. These concretions were glued together in one mass by the viscid bile, and probably formed the

obstruction which prevented the gallbladder from emptying. After the gallbladder collapsed, it was seen that the apparent bilobular condition was really due to a diverticulum. Cultures of the bile from the gallbladder remained sterile.

The patient made an excellent recovery. The morning after the operation, he showed a slight euphoria. This soon disappeared, and no other signs of encephalitis remained. The patient, however, had no recollection of the twenty-four hours prior to the operation, nor of going to the operating room. A few days after the operation, he stated that for six or seven months before he had had no energy, had been steadily losing in weight, had been forcing himself to do his usual routine work, and had derived little pleasure from his summer vacation.

Case 4.—*Clinical History.*—Miss B. S., aged 31, seen Sept. 4, 1923, had been ill for five weeks with a sore throat. Since that time she had been weak, extremely tired, restless, and had had considerable difficulty in sleeping. For two weeks she had had shooting pains in the legs and abdomen. She had also had a good deal of visual disturbance, disturbing dreams being common both day and night; on one day, for a period of a few hours, she saw only half of various objects and of the people about the house; there had been no diplopia. She complained of jerking of arms and legs, and of inability to think and concentrate. She was extremely nervous and fearful of some impending calamity.

Examination.—Examination revealed a rather restless, weakly woman, with flushed face, neck and chest; the thyroid gland was slightly enlarged; the pulse rate was 88. The tonsils were enlarged and red, but no pus was evident. The lungs were normal. The abdomen showed marked rigidity and extreme tenderness all over the upper right rectus, but no other signs. The abdominal reflexes were absent on the right side, the knee jerks exaggerated. There was a marked, continual jerking of the arms, legs and shoulders; tremor was present in the tongue and in the abdominal muscles. The temperature reached 101 F. on one occasion. The leukocyte count at first was 13,400; two days later it was 11,500. Spinal puncture was not made. Duodenal drainage showed the presence of some bile in the stomach; the *A* bile showed a moderate amount of pus and much mucus; *B* bile was not obtained.

Course.—Operation was performed on September 17 by Dr. Will Fisher. On opening the abdomen, the gallbladder pushed out through the incision; it was enormously distended and tense; the cystic duct was sharply flexed and bound down by adhesions, producing an obstruction at that point. The gallbladder was removed, and a drainage tube inserted. The bile in the gallbladder was thick, viscid, greenish black, with a considerable sediment but no stones. A culture was sterile. The patient made an uneventful recovery; all the symptoms of encephalitis, including pains, exhaustion, sleeplessness, tremor and jerking of the muscles disappeared promptly. She left the hospital at the end of two weeks, and has remained well.

Case 5.—*Clinical History.*—H. H., a retired druggist, aged 70, who was seen in consultation with Dr. W. R. Stephens, had been taken sick about Sept. 15, 1923. He complained of drowsiness and of shooting pains in the trunk and down the course of the right sciatic nerve. There was some mental disturbance, particularly impaired memory and lack of ability to concentrate; he was beginning to falsify about little things. On a number of occasions, he evidently had had double visions, for he expressed himself now as "not seeing things twice any more." He had troublesome dreams, and at times was irrational and held in bed only by restraint. Some peculiar swelling developed in the right side of the neck; it was opened by the attending surgeon, but only a little serum was

obtained. His usual weight had been about 220 pounds (99.7 kg.), and he had lost approximately 50 pounds (22.7 kg.) by November 1, when I saw him.

Examination.—Examination revealed marked stupor; though the patient could be aroused, he fell promptly into a deep sleep when undisturbed. There were marked quiverings and twitchings of the arms and legs, with tremor of the tongue. No evidence of the mass in the neck was present, except the small scar of the incision. The lungs were normal; the heart was slightly enlarged to the left, the tones variable, the pulse rate 64, blood pressure 110 systolic and 60 diastolic. The abdomen showed a slightly enlarged liver, with considerable rigidity, and, even in the drowsy state, some tenderness under the right costal border. The right upper abdominal reflexes were not obtained; the knee jerks were exaggerated, and a Babinski sign was present on the right. The urine showed albumin 2 plus, but was otherwise negative. The blood count showed 11,000 white cells. The feces were clay-colored. Blood chemistry showed urea 20.5 mg. per one hundred cubic centimeters of blood; creatinin, 3 mg.; blood sugar, 0.12 per cent.

Course.—Operation was performed by Dr. Will Fisher, November 1. The gallbladder was found to be distended, constricted in the middle and filled with stones varying in size from that of a pinhead to one-half inch (1.2 cm.) in diameter. One stone was entirely enclosed by the gallbladder wall. Thick, viscid bile was present in the gallbladder. The patient died within twenty-four hours after the operation, from cardiac exhaustion.

CASE 6.—*Clinical History.*—Mrs. S. N., aged 33, who was seen April 23, 1921, had complained for about a week of a sense of fulness in the right ride of the face, disturbance in taste, double vision, some discomfort in her right eye, insomnia and a tendency for the right arm and leg to go to sleep. The main findings on examination were an unblinking right eye, although on effort the eye could be closed; fulness of the right side of the face with lips drawn slightly to the left; pupils normal; the right knee jerk much increased; no disturbance in sensation except some anesthesia over the right side of the face. The condition cleared up in about ten days and was followed by a marked increase in appetite. I considered the case an abortive type of encephalitis.

In the summer of 1923, attacks of upper right abdominal colic began; they varied in intensity; two were so severe as to require morphin. The last severe attack occurred on Dec. 26, 1923. On Jan. 19, 1924, the patient was seen at her home, and the following history was obtained: For about a month she had noticed progressive weakness; she was dizzy, and most of the time she felt sick. Her complaint was of inability to raise the head, loss of control of the feet, inability to stand alone and jerking of the legs.

Examination.—Examination revealed a patient in a drowsy stupor. There was bilateral ptosis; on raising the eyelid photophobia was marked, and diplopia was then complained of; bilateral nystagmus was present. The temperature was normal; the pulse rate was 78; there was a marked tremor of the tongue; the neck was slightly rigid; the upper right abdominal reflex was not obtained, the others could be obtained with difficulty; there was no change in the reflexes of the extremities. The leukocyte count was 8,400. The urine showed a trace of albumin, but was otherwise normal.

Course.—Operation was performed on January 21, by Dr. Fred Douglass. The gallbladder was found adherent, and contained fifteen stones varying in size from one-half inch in diameter to that of a pinhead. A considerable amount of fine gravel was also present. Cultures were taken, but no results were obtained.

The patient made a quick recovery; there was great improvement in all the symptoms, although for a few weeks there was still a slight muscular weakness of the eyes, which necessitated a temporary change in her glasses. At the end of five weeks, the eye condition had improved so that she was again enabled to wear her previous glasses. Up to the present date, she has remained in perfect health.

Comment.—In the cases here reported, recovery from the encephalitis was so prompt after operation that we are justified in assuming an association between the biliary tract infection and the encephalitis. All of the patients have been seen since the operation. In none have any of the sequelae so common to encephalitis developed.

I have seen a few cases of encephalitis in which treatment by nonsurgical duodenal drainage produced good results. These are few, however, and it is my impression that there is usually definite obstruction to drainage of the gallbladder, and that treatment must be surgical.

CASE 7.—E. E., a school girl, aged 10½, seen with Dr. Ellis Kelly, July 2, 1924, had been taken sick on May 15, 1924, with fever, sore throat, vomiting and pain in the abdomen. The attack lasted one week. Ten days later, a second attack of fever and vomiting occurred. This time the vomitus consisted mostly of bile; the liver became enlarged, and definite jaundice, with highly colored urine and clay-colored stools, was noted. On subsidence of the acute attack, a subfebrile temperature persisted. The child complained of pain in the back and neck, with tenderness all over the body and of the feet. She was extremely tired and dizzy, and had some loss of memory. She said that on several occasions, when out in a bright light, she saw double. The father had noted some speech difficulty, and also loss of voice and hoarseness since the first attack.

Examination revealed a rather large, obese child, who appeared drowsy. The pupils reacted slowly but equally, and there was no muscular incoordination; there was a nystagmoid movement both to the right and to the left. The tongue when protruded moved a trifle to the left. The throat was clean, but there was inability to raise the soft palate. The pharyngeal reflex was absent. The thyroid gland was not enlarged; the heart outlines were normal, rate 90, tones clear, no murmurs. There was marked rigidity of the upper right rectus muscle, and the liver was enlarged three fingerbreadths below the costal border, and was tender. The abdominal reflexes were exaggerated, as were also the knee jerks. A right-sided Babinski sign was noted. The blood count revealed 3,929,000 red cells and 12,650 white cells. The urine examination was negative.

The subsequent history is not exactly clear, but as nearly as I can find out, the child went on to a state of marked asthenia resembling paralysis. All of the extremities and even the neck were supposed to be paralyzed. Three different diagnoses were made by various physicians: Sydenham's chorea, encephalitis of a choreic type, and poliomyelitis.

Comment.—In view of the recovery with absence of any residual motor phenomena, poliomyelitis can be excluded. I believe that the diagnosis of encephalitis is the most logical one, especially since the only other diagnosis made was chorea. This indicates that the clinician recognized an involvement of the nervous system.

A number of cases showing definite association between gallbladder infection and encephalitis in other ways may properly be included in this report. In the first two cases of this group, encephalitis occurred first and later gallbladder colic appeared.

CASE 8.—Mr. G. Z., a typesetter, aged 29, on Dec. 28, 1920, began to suffer from severe shooting pains in the great toe and calf of the left leg; the condition continued for ten days and then shifted to the inner side of the thigh, up to the gluteal fold, and then to the lumbar region. The pains were lightning-like, leaving no soreness. He had never been sick before. The personal and family history was of no significance.

The general examination at this time revealed nothing to account for this pain. After two weeks of suffering, however, it was noticed that the patient was drowsy. When questioned, he admitted having seen double for a number of days. Ptosis of the right eye developed. General examination revealed no changes in the reflexes. However, the drowsiness and the eye changes peristed for a period of several weeks. Convalescence extended over a period of two or three weeks longer.

This was undoubtedly a mild attack of encephalitis, with symptoms limited to lethargy and changes of the eye.

June 10, 1924, the patient was awakened with intense pain in the upper part of the abdomen, radiating to the back. He vomited three or four times, the vomitus containing bile. The attack lasted six hours. Following the attack, he had some indigestion, and found that he was unable to eat as freely as previously; otherwise he was comfortable. On July 28, he had a recurrence of the attack. He was seen at this time, and jaundice was present. There was marked soreness in the upper right quadrant of the abdomen, and a distinct mass, extending two or three fingerbreadths below the right costal border, could be made out. The urine contained bile.

CASE 9.—Mrs. E. A. H., aged 35, first seen March 17, 1921, when she had severe pain along the course of the left sciatic nerve; the pain was intense and not relieved by medication. It persisted for two weeks, when she became drowsy, complained of extreme fatigue, nervousness, a sense of tingling along the left forearm and leg, quivering of the arms and legs, jerking of the abdominal muscles and vivid dreams. She entered the hospital on April 2, 1921. The drowsiness increased. On April 6, she complained of double vision, which persisted for a week. Unilateral vision also was noted. Ptosis of the left eyelid then developed; photophobia was complained of, and a slight paralysis of the tongue appeared. Her temperature reached as high as 99.8 F.; the leukocyte count was 11,000. The duration of the attack was thirty-one days. During the spring of 1924, she had several attacks of severe upper abdominal pain, which the attending physician, Dr. Frank C. Clifford, thought might be either acute gastritis or, more likely, gallstone colic.

CASE 10.—Miss M. R., a district nurse, aged 41, was seen first in 1917, during an attack of gallstone colic which persisted for ten days. The gallbladder was drained, and stones were removed. March 1, 1921, the patient was seen at the office. For eleven days, she had become fatigued easily, had been unable to do her work, her memory had been deficient, and she had often skipped patients in her rounds. She complained of generalized muscular aching and hypersensitiveness of the skin. She rested in bed for several days, and then felt better. After getting up, she again became exhausted, and was then sent to a hospital. There she developed double vision, ptosis of both eyelids, marked drowsiness, remark-

ably vivid and beautiful visual dreams, and finally became extremely somnolent. The patellar reflexes were increased; the adbominal reflexes on the right side were absent; the Babinski and Kernig signs were not obtained. The leukocyte count was 9,900. The duration of the entire attack was forty-three days.

This patient had distinct parkinsonian symptoms following the encephalitis. They were of mild type and eventually cleared up, but she was unable to work for a year after recovering from the encephalitis. This case is one of a known infection of the gallbladder, in which the gallbladder was not removed; later, the infection was followed by encephalitis with long drawn out convalescence.

COMMENT

I believe it is worthy of serious consideration that such a large proportion of my cases of encephalitis have been associated with gallbladder disease. It is rather difficult not to believe that there is more than an accidental association between the two conditions. It is significant, too, that some of the patients seen before I recognized this association should now present the pathologic condition associated with gallbladder disease.

That encephalitis could be due to a focus of infection located in the gallbladder and biliary tract seems at first, perhaps, too radical and unorthodox to be easily accepted. It might be worth while, therefore, to consider briefly some of the objections that might come to mind.

The syndrome observed in these cases is unquestionably that of encephalitis. But, is it epidemic encephalitis? This can be answered definitely in the affirmative. In the older books on nervous diseases published prior to the recent influenza epidemic, encephalitis is scarcely mentioned. Church and Peterson, in the fifth edition of their book, devote less than two pages to the subject. Dr. Church's remarks are of extreme interest and importance. He says: "The writer has seen two cases, one following influenza, and the other after pneumonia; and one case with some unknown infection associated with acute nephritis. *It is a matter of speculation whether various infections act locally or by their elaborated toxins. An interval, a sort of incubation period, is often noted between the infectious disease and the manifestation of the cerebral symptoms.*" (The italics are mine.)

Since so few cases were reported prior to the influenza epidemic, all cases seen since that time must belong to one group and be classified as epidemic (lethargic) encephalitis, variations in type being due to the protean nature of the disease and to differences in severity such as is observed in all diseases, regardless of whether they occur in large numbers (epidemic) or in occasional cases (sporadic); similar variation is marked in anterior poliomyelitis, a disease having much in common with encephalitis.

Why are no more cases of diseased gallbladders reported in connection with encephalitis? Study of the literature shows that the

necropsy material is inadequate. In a large general hospital, the records for three and a half years showed not one complete necropsy in a case of encephalitis; naturally, the clinical history recorded was not of great value in checking up the possible presence of gallbladder disease. The only complete necropsy reports I found in a fair though incomplete search of the literature were given by Tilney and Howe [2] in their book on epidemic encephalitis. They report two cases (Cases 5 and 11). Both show definite and marked gallbladder disease.

Meyer-Bisch,[3] on testing six cases of encephalitis with 100 gm. of levulose, found that five gave reactions for levulose in the urine, an indication that the liver function in these cases was impaired.

Books on medicine call attention to the fact that in jaundice, delirium, mania, convulsions, coma and death occasionally occur. These have been attributed by some to the toxic effect of bile, and by others (von Leube) to some metabolic disturbance. It seems to me far-fetched to attribute these symptoms of injury to the brain to the jaundice, because chronic jaundice of various grades of severity is frequently observed without such symptoms. When these serious symptoms occur, we must, therefore, assume that some other factor is present. The symptoms are not unlike those found in encephalitis of severe grades, and it seems much more reasonable to account for them by the presence of some unusual toxin in the biliary tract which has selective action on the brain.

Finally, attention must be called to Wilson's disease, in which peculiar changes are found in the basal ganglia, not unlike those present in the postencephalitic parkinsonian cases, accompanied by cirrhosis of the liver. This establishes a definite possibility for the occurrence of an association between liver disease and brain disease.

It might be mentioned in passing that certain drugs which are known to injure the liver also give rise to marked disturbance in brain function, with a clinical picture exactly like that in chronic jaundice. This is especially true for chloroform and phosphorus poisoning and for acute yellow atrophy of the liver.

The diagnosis of gallbladder disease or biliary tract infection is not easily made in these cases. This part of the clinical picture is not outstanding, and it will often require all the clinical skill of the trained internist, with the use of every laboratory procedure known to be of value in the diagnosis of gallbladder disease, to make the diagnosis. Only exceptionally does the presence of abdominal disease stand out clearly.

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2. Tilney, Fred., and Howe, H. S.: Epidemic Encephalitis, Ed. 1, New York, Paul B. Hoeber, 1920.

3. Meyer-Bisch, R., and Meyer, E.: Diabetes Insipidus, Ztschr. f. klin. Med. **96**:469 (Feb.) 1923.

The reason probably is that these cases are, so far as the gallbladder is concerned, cases of low grade infection, with the toxic picture predominating over the infectious.

Attempts at verification by animal inoculation will not prove successful, because bile cannot be used, and the exact toxin cannot be reproduced except under the exact conditions prevailing within the gallbladder itself.

In conclusion, I may mention that we are coming to realize more and more the frequency with which gallbladder disease occurs in early life. The recent contribution of Mann and Willson [4] is especially illuminating on this point.

1708 Jefferson Ave.

4. Mann, A. T., and Willson, H. S.: Chronic Gallbladder Disease in Young Adult, J. A. M. A. **83**:981 (Sept. 27) 1924.

REACTIVE GLIOSIS IN A CASE OF BRAIN TUMOR *

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It is a matter of common observation that the cerebral hemisphere, that is, the seat of a new growth, is usually larger than its fellow of the opposite side. This enlargement is due to several factors, the first of which is the presence of the tumor in the involved hemisphere. In the second place, there are vascular disturbances which may account for some of the discrepancy. Edema, thrombosis and hemorrhage are all known to occur within or in the vicinity of a tumor. Reactive gliosis also plays a part in the notable enlargement of the involved hemisphere. There are cases in which the tumor is of small size, compact, sometimes even calcified, in which, in spite of the absence of vascular disturbances, the hemisphere in which it is situated is enlarged out of all proportion to the size of the neoplasm. It is in these cases that reactive gliosis is described to account for the phenomenon.

"In many brain tumors, especially the gliomas," writes Ewing,[1] "the entire brain tissue may be the seat of structural changes resulting probably from general disturbance of the circulation. These consist in increase of the glia nuclei especially in the deep cellular layers, disturbed polarity of the cells, partial or complete tigrolysis, shrinkage or complete atrophy of the cells, pyknosis, karyolysis or homogenization of the nucleus, and marked increase of pericellular nuclei." According to Merzbacher,[2] the glia reaction is most marked in cases of glioma and sarcoma, whereas metastatic carcinoma provokes but little reaction, and meningeal tumors stand midway in the scale. Spiller[3] says: "If the brain be examined by a neuroglia stain there will be found in some instances an overgrowth of neuroglia even in areas at a considerable distance from the tumor."

To the older writers this gliosis throughout the hemisphere containing the tumor was an established fact, but their observations depended

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* Presented before the Clinicopathologic Conference, Dec. 15, 1924.

* From Blackburn Laboratory, St. Elizabeth's Hospital, Washington, D. C.

1. Ewing: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, 1919, p. 383.

2. Merzbacher: München. med. Wchnschr., 1909, p. 2051.

3. Spiller: Am. J. Med. Sc. **47**:29, 1914.

largely on estimates as to the number of glia nuclei present in a given field or on the density of the fiber meshwork. To the eye of the observer, both of these are subject to variation according to the perfection of the stain, so that except in cases of gross increase there is some doubt left in the mind of the unprejudiced observer as to the validity of the observation. That there is a reaction on the part of the glia in the area near the tumor is an acknowledged fact, and that this reaction is

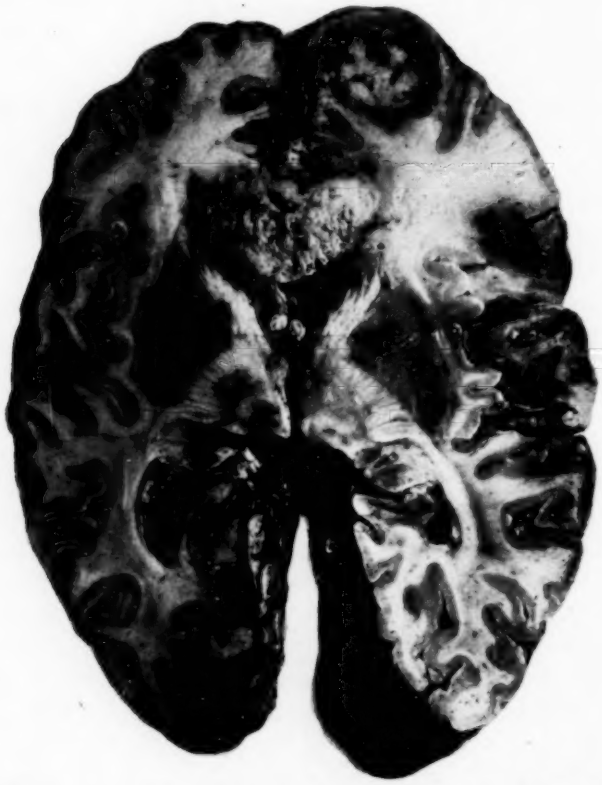

Fig. 1.—Gross view of brain showing gliomas in (*a*) frontal pole, (*b*) corpus callosum, (*c*) frontal operculum. Moderate enlargement of the left hemisphere is shown. The photograph was made from the under side of the horizontal section so that the sides are apparently reversed.

observable in all portions of the brain is postulated from the actual increase in size of one hemisphere over the other, but until the advent of the newer selective impregnation methods for the neuroglia, the estimate of the increase was always uncertain, depending as it did on the judgment and experience of the investigator.

An opportunity for a renewed study of the condition was recently presented in this hospital.

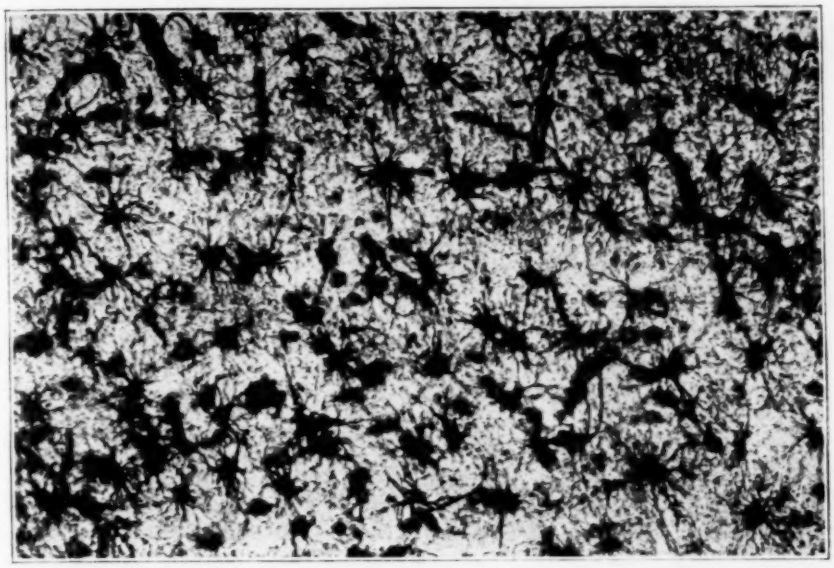

Fig. 2.—Reactive gliosis in left frontal pole in neighborhood of the tumor. Cajal stain; × 200. Compare with Figure 3.

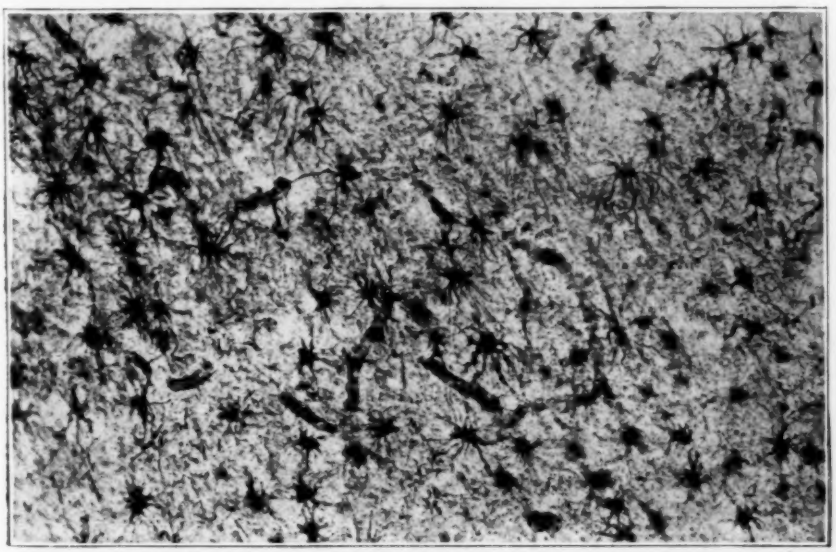

Fig. 3.—Neuroglia cells in right frontal pole. Cajal glia stain; × 200.

A colored man, aged 60, came to necropsy less than two hours after death. On removal of the brain it was found that there was a small tumor situated in the extreme frontal pole on the left side. Sections were immediately taken from symmetrical parts of the brain for fixation in the formol-bromid solution recommended by Cajal.[4]

The brain was then placed in 10 per cent. neutral formaldehyd saline solution. After fixation, longitudinal sections were made. They disclosed not only the original tumor in the frontal pole on the left side, but two other unconnected tumors, one occupying the genu of the corpus callosum and the other the frontal operculum on the left side. The tumors were circumscribed but not encapsulated, appeared to infil-

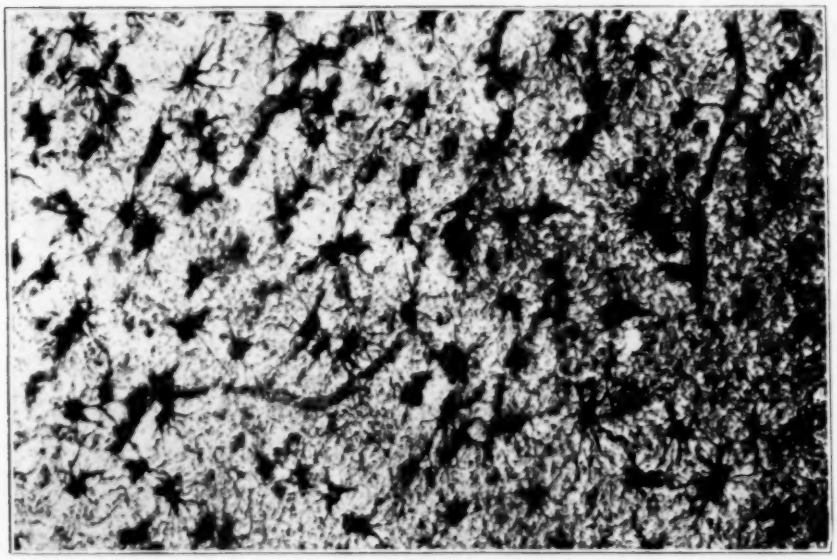

Fig. 4.—Reactive gliosis in left second frontal convolution; to be compared with same area on opposite side in Figure 5. × 200.

trate the surrounding tissue to a slight extent, were dark at the periphery and pale in the center. The whole left hemisphere was slightly larger than the right (Fig. 1). Microscopically, the tumors were found to differ slightly in histologic structure. The basis of architecture in the tumor of the frontal pole was the blood vessel. Every vessel was surrounded by large numbers of small oval nuclei arranged for the most part tangentially, in layers from 6 to 15 cells deep. The vessels were

4. For a review of this and other methods for selective staining of the neuroglia consult Bailey and Hiller: The Interstitial Tissues of the Central Nervous System: A Review, J. Nerv. & Ment. Dis. **59**:337, 1924.

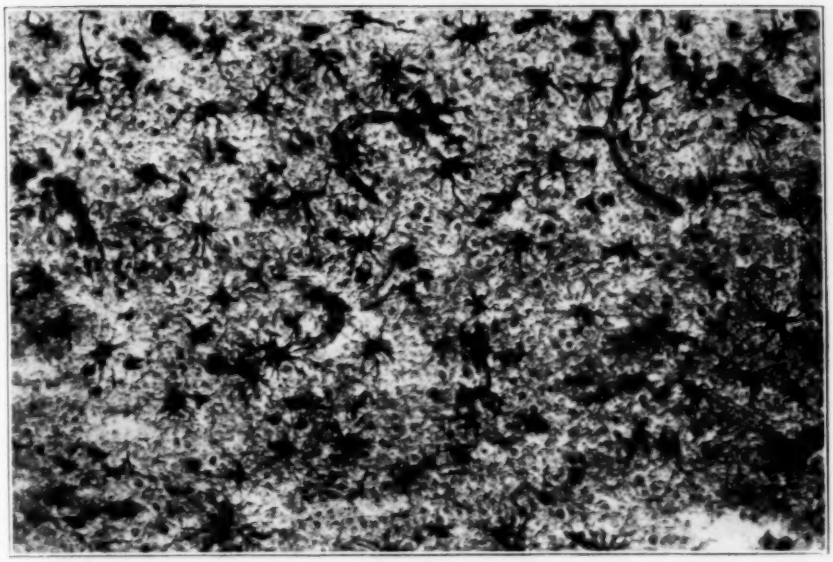

Fig. 5.—Neuroglia in right second frontal convolution. Cajal stain; × 200

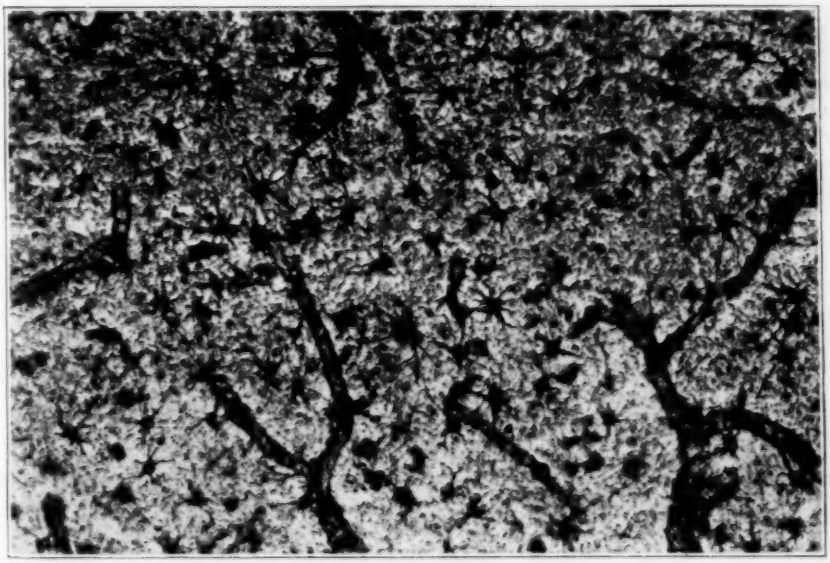

Fig. 6.—Neuroglia in left precentral convolution. Compare with Figure 7. Cajal stain; × 200.

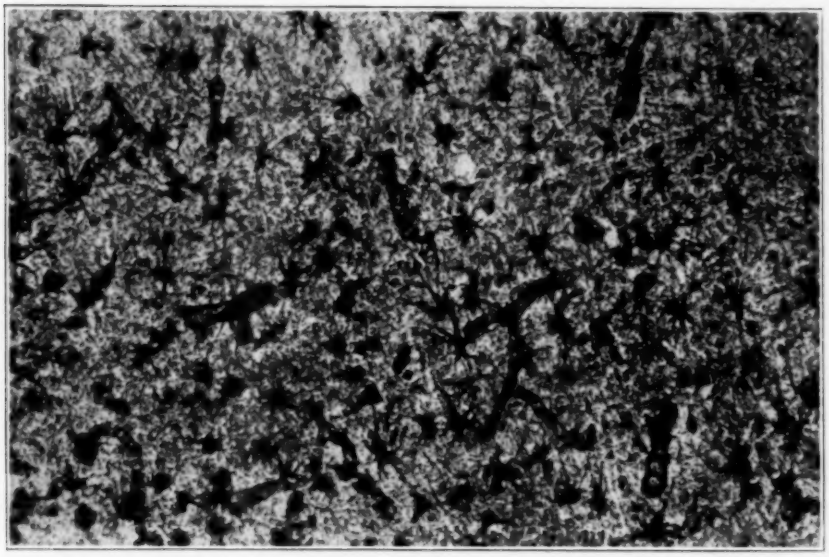

Fig. 7.—Neuroglia in right precentral convolution. Cajal glia impregnation; × 200.

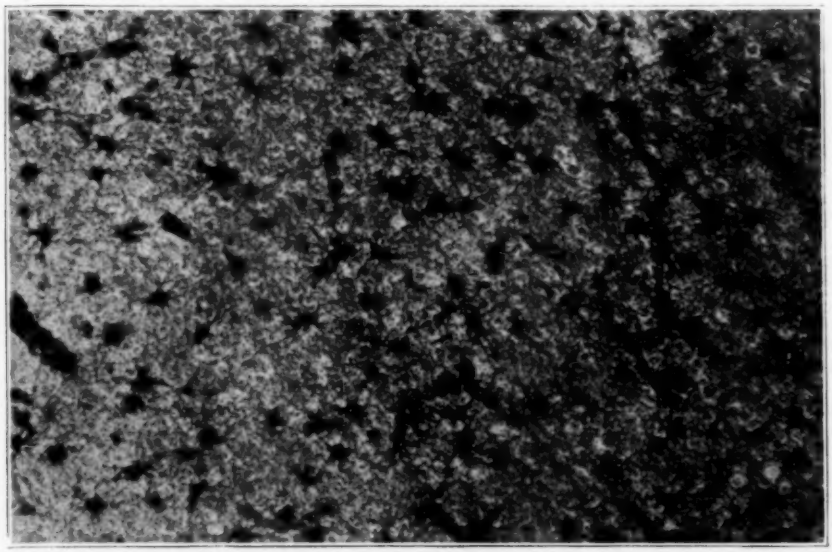

Fig. 8.—Neuroglia in left first temporal convolution. Compare with Figure 9. Cajal stain; × 200.

large, with well formed walls and hyperplastic endothelium, which could be distinguished from the tumor cells by the larger size and paler staining reactions characteristic of the endothelial nucleus. The vessels at some distance from the growth also presented collars of cells resembling in all respects the typical cells of the tumor. Fatty granule cells or products of degeneration were rarely present, and there were no signs of inflammation. In the more central portions of the tumor, the walls of the vessels became notably thickened and their caliber diminished, and within the pale area noted in the gross description there existed nothing but thrombosed vessels with cellular detritus and pale staining necrotic material. One vessel was observed to maintain its patency, and about it were grouped a large number of tumor cells.

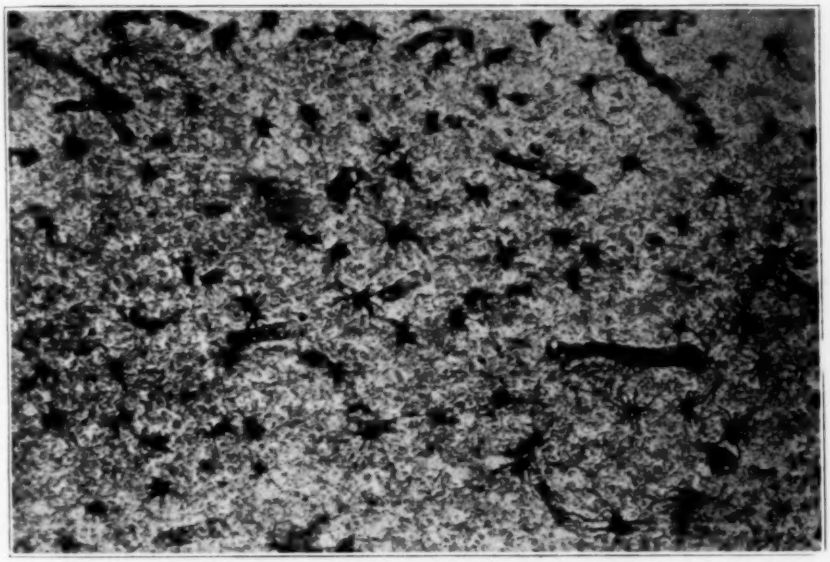

Fig. 9.—Neuroglia in right first temporal convolution. Cajal stain; × 200.

The other two tumors presented much more the appearance of the ordinary cellular glioma, although here also vessels were plentiful, sometimes varicose and often thrombosed.

The Cajal stains on the first tumor showed no glia fibers within the tumor mass, but at the boundary there were to be observed large numbers of large vacuolated cells with twisted swollen processes, individual processes separate from cells and large grotesque nuclei with indeterminate cell bodies. It was only at a short distance from the tumor proper that the reactive gliosis became evident in the manner shown in the photograph (Fig. 2). After seventeen days' hardening of the blocks originally fixed in the fomaldehyd-bromid solution, sections at 20

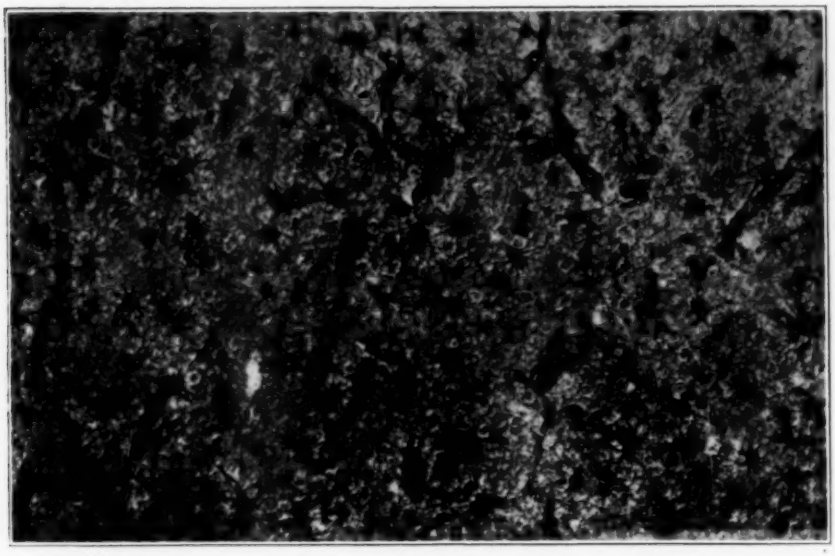

Fig. 10.—Neuroglia in left occipital lobe. Compare with Figure 11; × 200.

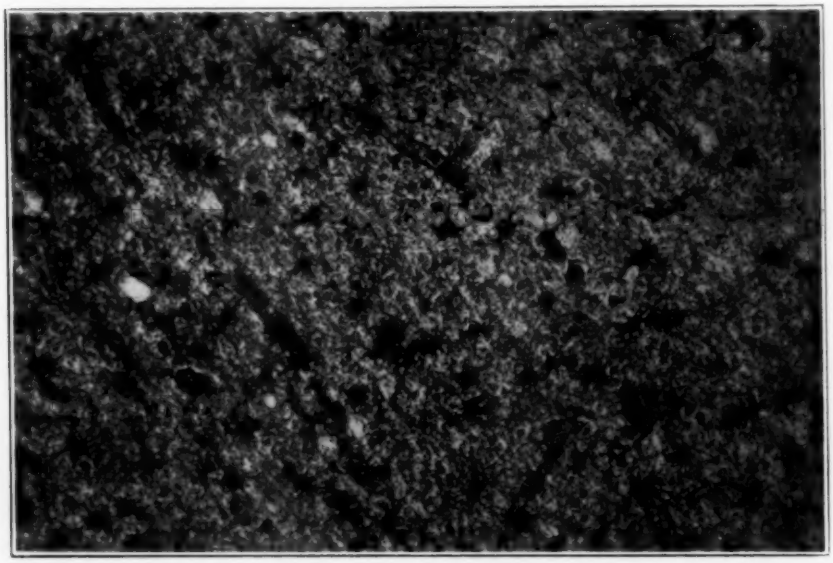

Fig. 11.—Neuroglia in right occipital lobe. Cajal glia impregnation; × 200.

microns were cut with the freezing microtome and impregnated by the gold-sublimate method devised by Cajal. The technical work was performed by Dr. Edna G. Dyar of this laboratory.

A photographic study was then undertaken. The photographic laboratory of the Army Medical Museum was placed at my disposal through the kindness of Major Callender and Dr. Kinney. Each slide was photographed under identical conditions of magnification and illumination. The field chosen in each case was at the boundary of the cortex and white matter, and an effort was made in each case to obtain the maximum number of glia cells within the range of the camera. The photographs were then taken, and the number of cells appearing in the 5 by 7 picture of each area was counted. Practically the only personal factor entering into this study therefore was the choice of the field. This was, I believe, a small one, for the distribution of the neuroglia at the cortico-medullary junction is remarkably regular. The count was made by both Dr. Dyar and myself, and varied within small limits.

The comparison in the number of cells on the two sides of the brain yielded the following results:

	Left	Right
Prefrontal	145	104
Frontal	136	96
Precentral	123	107
Temporal	119	85
Occipital	116	84

The hyperplasia of the neuroglia is particularly noticeable in the vicinity of the tumor, and here also a true hypertrophy is evident, the cells being large, with much cytoplasm and abundant processes. Figure 2 shows how great has been the actual increase in size in comparison with those on the opposite side (Fig. 3). The convolution shown in Figure 2 was not directly involved by the tumor.

The overgrowth in size of the neuroglia cells is less marked in the portions of the brain farther removed from the tumors; and in sections from the temporal and occipital lobes, no difference in size is appreciable, but the number of cells is always definitely increased on the side of the lesion.

SUMMARY

The opinion of the older writers that there is actual neuroglia overgrowth throughout the hemisphere, which is the seat of a glioma, is borne out by a carefully controlled photographic study on unimpeachable material.

PSYCHOSIS ASSOCIATED WITH TRAUMA OF THE HEAD

REPORT OF A CASE OF TRAUMA SUSTAINED IN INFANCY AND DETERMINING A LATER DEVELOPING DURAL TUMOR

A. E. TAFT, M.D. AND E. A. STRECKER, M.D.
PHILADELPHIA

Clinical instances of traumatic psychosis authenticated by necropsy findings are rare enough to justify a report. Even with the inclusion of doubtful cases not confirmed postmortem, it is likely that the incidence of well established psychotic syndromes remains less than 1 per cent. This stands in contrast to the fact that trauma of the head has come to be recognized as a factor of some importance in the production of juvenile delinquency. Healy [1] places the determining influence of head trauma in this group at about 3.5 per cent., and states that "a larger number of head injuries are to be found among the delinquents than among nondelinquents." Strecker and Ebaugh [2] found that thirty cases of striking behavior disorders in a group of about 600 delinquent children (5 per cent.) were due to cerebral trauma. However, the evolution into a frank psychosis in adult life is seemingly an unusual phenomenon. Eager [3] reported from his experience as Chief of the Lord Derby War Hospital at Warrington that 1.75 per cent. of mental disease followed injury of the head. In his civilian hospital experience, he estimated the psychotic incidence associated with trauma of the head at 2 per cent.

REPORT OF A CASE

The subject of this report died at the age of 68, the last thirty-six years of her life being spent in a hospital for mental disease. Her psychosis suffered at the hands of changing psychiatric nomenclature. For many years, it was designated "chronic mania"; later the opinion veered to dementia praecox, probably following the announcement of Kraepelin that many examples of chronic mania were in reality dementia praecox. The early history is interesting; but, unfortunately, from the point of view of diagnosis, it was not obtained until after the pathologic findings at necropsy had sufficiently stimulated scientific curiosity.

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* Read before the Philadelphia Neurological Society, Jan. 23, 1925.

1. Healy, William: A Review of Some Studies of Delinquents and Delinquency, Arch. Neurol. & Psychiat. **14**:25 (July) 1925.

2. Strecker, E. A., and Ebaugh, F. G.: Neuropsychiatric Sequelae of Cerebral Trauma in Children, Arch. Neurol. & Psychiat. **12**:443 (Oct.) 1924.

3. Eager, Richard: J. Ment. Sc., April, 1920.

History.—The salient historical points are quoted: "My sister was born in 1856, sixty-eight years ago, and it is difficult for one to recall after a lapse of so many years the full details of her infancy. However, when an infant in arms, her nurse let her fall, and evidently the fracture of the skull happened at that time. Immediately, or shortly after the fall, both eyes crossed. That was followed by brain fever. After recovery from the fever, she was paralyzed on the left side, causing her to drag her left leg more or less, and her arm and hand were rendered almost useless. Every summer, she was taken to the shore or the mountains with the hope that the change would be beneficial, but she never recovered the normal use of her left side. At no time was her articulation impaired. She was of a buoyant temperament and very social in her disposition. I do not remember about her school work, but I think she was probably about the average pupil. As she grew older, say in the late twenties or early thirties, she became very self-willed, and it was impossible to control her at times, so we were obliged, for her own good, to place her under restraint in hospitals for mental troubles. She first went to the A——— Hospital in 1888, and would be brought home from time to time with the hope of being able to control her, but invariably there was a breakdown after a few months and she would have to return to the hospital. This continued until Aug. 11, 1903, when I was obliged to go to Cape May and bring her to the B——— Hospital, under whose care she remained until her death."

Piecing together various bits of information from the usual inadequate hospital record of three decades ago, it appears that the patient originally and fairly early in life manifested the signs of a so-called traumatic constitution, since she "could not be controlled, was restless, insubordinate and remained out late at night." There was no evidence of mental defect. During the major part of her hospital residence, there were episodic excitements lasting several months and spaced by shorter intervals of comparative freedom from marked mental symptoms. When disturbed, there were to be seen increased vasomotor and psychomotor activity, distractibility, disassociation, incoherence and suspiciousness. Occasionally there was euphoria. At these times, the patient tended to be seclusive; was quite noisy and abusive, threatening, profane, obscene and untidy. There were no definite hallucinatory-delusional trends. However, from time to time her behavior indicated that she believed that she was one of the nurses. Once she insisted that she had been removed from the hospital against her will, anesthetized and subjected to an operation. Again she described snakes which she had seen swimming in a natatorium pool. It will be seen that many symptomatic elements bespoke an illy defined circular psychosis, or at least a series of manic attacks, but some of the criteria of schizophrenic excitement (disassociation, suspiciousness, etc.) were not wanting. When approachable for direct examination, there was good retention and memory, orientation, fair school knowledge, imperfect calculation ability, defective knowledge of current events, no sustained hallucinatory or delusional trends and limited insight.

Physical Examination.—The physical record of the patient deserves consideration. The findings in several neurologic examinations were not consistent. A slight internal strabismus of the left eye and sluggish pupils were noted. Several times the left patellar reflex was quite active, while the right was absent, as was also the Achilles reflex. The left foot dragged a trifle on walking, and there was some diminution of power. The left arm was awkward and weak. Six years before examination, the patient suddenly became unconscious and fell. A physician, called to the ward at 9:30 a. m., "found the patient lying

on the floor, unconscious, with flushed, but not cyanosed face, stertorous breathing, tongue slightly protruded, moving back and forth in slow rhythmic movement, a sort of chewing movement with her jaws. There was no convulsion or twitching to be seen at this time. The eyes were shut, but easily opened, and the pupils were equal and contracted, not reacting to light. The Babinski reflex was marked on both sides. The nurse's report said that she was sitting on the bench, was heard to cry out, and fell, striking her head against the table. One attendant said that she seemed to be having a convulsion. There was a small bruise on the side of her forehead the size of a twenty-five cent piece, and a small scratch across the bridge of her nose. The unconsciousness lasted about ten minutes; after that it was partial for a short time." There were no effects of this attack manifested at the end of twenty-four

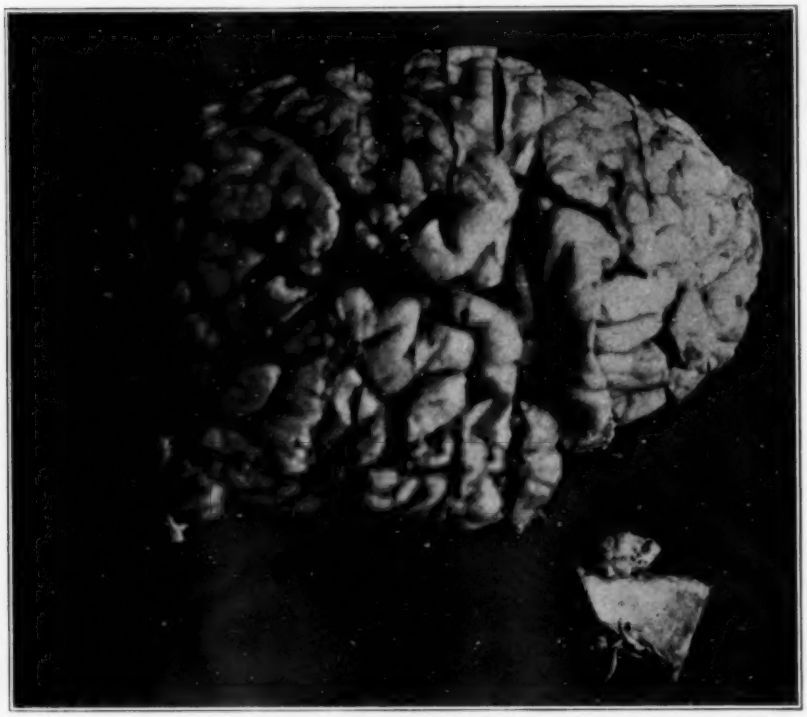

Fig. 1.—Lateral view of right hemisphere showing defective gyral pattern at point underlying the tumor. Below is a fragment of dura with tumor attached.

(The illustrations are not photomicrographs, but negatives of slides done with Weigert stain. Consequently, the fiber portions are light and the cellular parts are dark.)

hours. The patient had at least two attacks of cholecystitis—one in February, 1923, and one recently, which caused her death. Her blood pressure was never above 142. The urine was generally negative, except for one analysis in which there was a heavy cloud of albumin and many red blood cells. The blood count was within normal limits, with of course a leukocytosis (25,800-21,600) during the acute gallbladder episodes. The Wassermann test was negative.

SUMMARY

To summarize the points of clinical interest: The patient sustained a fractured skull in infancy, and either it alone or in conjunction with the so-called "brain fever" (meningitis?) resulted in a partial left hemiplegia. At the end of the third decade of her life, her personality changed and she began to behave in a manner which, in the light of the injury of the head, suggested the likelihood of the development of a so-called traumatic constitution. At about this time she was placed in a hospital for mental diseases, and for at least twenty-three years had a psychosis marked by manic-like symptoms, but with enough disintegration to suggest dementia praecox. During all these years, there was but a single reliable objective phenomenon, namely, the "convulsion" six years before, which might have brought the head injury into consideration as an etiologic factor.

Pathologic Findings.—The following note was made by Dr. C. B. Farr at the postmortem examination.

Skull and Brain: On removing the scalp, there was a depressed, irregular area 8 cm. in length and 1.5 cm. in width, just above the right temporal muscle, its lower border conforming to the insertion of this muscle. The inner surface of the skull presented a somewhat similar appearance, but in the center of the area there was an elevated and extremely rough projection the size of a nickel. There was extensive calcification of the dura along the superior longitudinal sinus. The cortex at the lower portion of the fissure of Rolando, corresponding with the elevated area in the skull, presented a depressed area the size of a thumb nail with surrounding inflammatory thickening of the membranes.

Spinal Cord: The spinal cord was not removed.

Brain (by A. E. Taft): The specimen was a good sized brain. The pia was cloudy throughout and showed a moderate degree of edema over the convexity on the right side; much less over the left. The pial vessels were injected. At the posterior extremity of the first frontal convolution, there was what appeared to be a cyst of the pia-arachnoid. On removal of the membrane, this was found to be due to an anomalous convolutional pattern appearing as a reduplication of the operculum in very narrow gyri (Fig. 1). This formed a space at the anterior end of the sylvian fissure, which is not infrequently seen in defective development with lack of closure of the fissure, thus leaving the island of Reil partially exposed. Over this area the superficial pial vessels were large and numerous, suggesting a possible early circulatory disturbance with consequent numerous anastomoses. The frontal lobes, including the central region, presented a moderate degree of atrophy. The gyri everywhere were of fair breath and average complexity. The right frontal lobe had four anteroposterior convolutions; on the left, there were but three. The basal vessels were tortuous, dilated and showed a considerable degree of atherosclerosis. This was particularly marked also in the middle meningeal vessels of both sides, especially in those on the right, which were somewhat larger than those on the left. There was no gross change in the cerebellum, pons or medulla.

On the inner surface of the dura, at a point about corresponding with the lower extremity of the central fissure, was a small, sharply circumscribed tumor, 2 by 1.25 by 1 cm. in diameter (1 cm. represents the thickness). The

surface was indefinitely lobulated and cross section showed a small central calcified area with the surrounding tissue somewhat striated, grayish white. The consistence of the tumor was firm but not hard. The surrounding underlying dura was slightly increased in thickness.

Frontal sections of the gross specimen of the brain revealed a small area of degeneration situated at the lateral angle of the anterior horn of the right lateral ventricle, at the level of the pulvinar (Fig. 2). There was a loss of tissue about 0.5 cm. in diameter crossed by strands of delicate fibers. This did not appear as a focal softening, but more nearly resembled a small porencephalic defect. It apparently represented the location of the fibers destroyed by the atrophy of the cortex described above.

In addition to this, the right side of the adjacent corpus callosum was thinner than the left side.

Fig. 2.—Frontal section through right hemisphere, showing small porencephalic lesion.

There was also a small softened area in the left putamen which appeared to encroach on the fibers of the internal capsule.

Microscopic Examination.—Sections from twenty-nine areas of the brain cortex were examined, including eight from the area underlying the tumor.

Moderate edema was seen in sections over the vertex. There was notable cell thinning in the cortex in nearly all areas, corresponding in degree with the arteriosclerosis present, which was most marked in the smaller vessels and the capillaries, both of the pia and within the brain tissue. The greatest reduction in cortical cells was present in the sections from the area of narrowed gyri adjacent to the tumor. Here there was a variance from marked cell loss, with shrinkage of the few remaining cells, to complete replacement for the depth of the cortex by a mat of glia fibers. A diffuse increase in glia cells

in the white substance was generally present, with marginal glia fiber overgrowth, and many amyloid granules. Satellitosis was present in a moderate degree in some areas.

The changes in the essential nerve tissue varied in degree in the different areas, and were more pronounced in the right hemisphere than in the left. A considerable degree of vascular change was present throughout.

In the right caudate nucleus and putamen of the lenticular nucleus were multiple, small, softened foci. In the larger of these there was complete tissue degeneration with cyst formation.

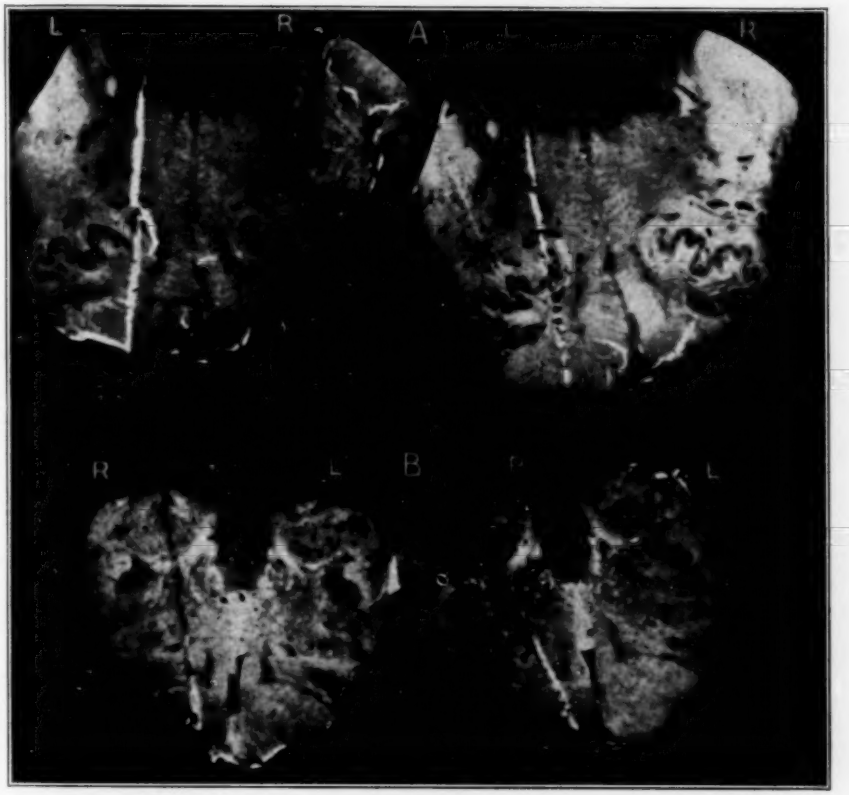

Fig. 3.—Duplicate sections through two levels of the medulla showing pyramidal tract on the right smaller than that on the left, and narrowing of the olivary band adjacent to the smaller pyramid.

In the right subthalamic region, adjacent to the substantia nigra, there was a microscopic hemorrhage into the tissue. As elsewhere, there was considerable arteriosclerosis, with occasional perivascular lymphoid cells and phagocytes in small numbers.

In sections through the medulla, the pyramidal fibers on the right side were much reduced in number, the bundle appearing about half the size of that on the left (Fig. 3). The adjacent portion of the superior olive was notably changed; the nerve cells of the olive were rarely present in this part, and there was considerable glia replacement, both cellular and fibrillar.

The tumor was made up of cellular and fibrous parts, which were associated in varying proportions in different parts of the growth. The cellular portion showed whorl formation, and there were numerous calcified concretions showing concentric arrangement. The vascular supply was fairly abundant.

The tissue showed the characteristics of meningeal endothelioma.

COMMENT

In evaluating these findings and interpreting them in relation to the clinical signs, it is necessary to differentiate the age changes associated with arteriosclerosis and those associated directly with the early injury.

One may count the diffuse cell thinning and glia increase, as well as the focal softenings, as secondary to vascular sclerosis. The convolutional anomaly and the tumor, with the overlying endostosis, are to be reckoned as having a distinct relation to the injury and the later developing nervous and mental symptoms.

The narrowed gyri and their limited cell content would appear to have two causal factors: the stage of development at the time of injury and the later irritation (pressure?) of the overlying meningeal and bony tumors.

Recent literature yields little to show that meningeal tumors have been found and studied in relation to previous injury of the head with later developing mental disorder. Cushing [4] reports a case, with histologic study, in which symptoms developed twelve years after injury. Mental disorder was not apparent. Spiller and Kirkbride [5] were the first to report in the literature the occurrence of meningeal tumor associated with bone changes. In a study of a group of dural tumors, Cushing [6] concludes that they arise from the arachnoid, and that the bony tumor (endostosis) is secondary to the meningeal growth. He gives them the name of meningiomas.

Mallory's [7] painstaking and exhaustive cytologic study indicates that these growths are not endothelial in origin, as has been considered. From embryologic theory, it is found that the dura and the arachnoid develop up to a certain point as one membrane. At the time of their separation, their apposed surfaces, which bound the subdural space, are not lined with endothelium, but only by fibrous tissue cells. He therefore suggests the name arachnoid fibromas. He says further of these tumors that they may grow rapidly or slowly, and that they contain numerous cellular and fibrous whorls or none at all. They derive their

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4. Cushing, H. W.: Cranial Hyperostoses Produced by Meningeal Endotheliomas, Arch. Neurol. & Psychiat. **8**:139 (Aug.) 1922.

5. Spiller and Kirkbride, in discussion on Cushing (Footnote 4), Arch. Neurol. & Psychiat. **8**:152 (Aug.) 1922.

6. Cushing, H. W.: Brain **45**:282 (Oct.) 1922.

7. Mallory, F. B.: J. M. Res. **41**:349 (March) 1920.

blood vessels and the connective tissue of their stroma from the dura. They can invade the dura, the skull and the soft parts outside, but they cannot pass through the arachnoid, from which they arise and invade the brain.

DISCUSSION

DR. WILLIAMS B. CADWALADER: Dr. Strecker said that in this case there had been found a tumor implicating the motor cortex. Last spring Dr. Spiller called my attention to the fact that he had seen a report of an endotheliomatous tumor growing on the motor cortex without having produced jacksonian epilepsy. Since then I have examined a number of the specimens at the laboratory at the University in an attempt to estimate the frequency of such occurrences. There were few instances, I think four in all, in which a tumor had implicated the motor cortex and had not been associated with jacksonian epilepsy. In Dr. Strecker's case there had been two convulsions, but from his description they were bilateral general convulsions, not jacksonian in character. Furthermore, he stated that the microscopic examination of sections taken from the cortex which had been compressed by the tumor mass had shown considerable destruction of the cortical cells. It may be that the nonoccurrence of jacksonian epileptic attacks could be explained by assuming that the destruction of cells was so great that they were no longer capable of being irritated by the presence of the tumor. It strengthens my belief that in some cases a tumor may implicate the motor cortex without producing focal epilepsy. Therefore the absence of jacksonian attacks need not in all instances indicate the absence of a tumor.

SIGNIFICANCE OF "ACCESSORY" BEHAVIOR ACCOMPANYING VOCAL REACTIONS*

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The method of observation of human behavior that I propose to present and discuss is part of an attempt to observe normal and pathologic human behavior from an objective point of view, and to bring meaning into a whole group of activities that we unscientifically call "casual." A necessary assumption, if we are to have a science of psychology, is that all behavior can be completely expressed in terms of (1) the antecedents of an organism, (2) its present physiologic state, and (3) the stimuli acting on it. If we had sufficient data, we should be able to understand completely all responses or reactions of the organism. Moreover, a quantitative relation between stimulus and response could be established if the exact state of the organism were known at the moment at which a stimulus was given. In other words, an animal will always give exactly the same response when it is confronted by the same stimulus, provided its general state is the same. If its general state has changed, the response will vary with this change in a way that is predictable. I include under antecedents of the organism, the ordinary processes of the individual's growth and the environmental experiences which have become a structural or functional part of the organism (habits, "memories," etc.). Under the physiologic state of the organism, I include the general state of health, its relative passivity or excitability, its readiness for response (in terms of immediately preceding experiences), the state of the viscera, skeletal musculature and central nervous system. A great number of alterations in the environment or in the body economy of the animal present themeslves without having sufficient strength either in intensity or duration to cause discernible response. Slight variations in temperature, moderate air currents, vague noises, people on street cars, automobiles passing the house—all these belong to a large group of stimuli that do not approach the level of physiologic or psychologic excitability, and hence may be called *non-effective*. In this paper, I deal only with environmental or situational changes sufficient in intensity or duration to become *effective* stimuli. If we subject an organism to an effective stimulus, we may expect a

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defined response which can be said to be the sum or result of past experience, present physiologic state and the impinging stimulus. If this be true, this response is the only possible response. Obviously, it is without point to speak of maximal or minimal or equal response if only one response is possible. On purely theoretical grounds, we must accept the reaction of the organism to stimulus as following an all-or-none principle, because if the stimulus be effective, only one result can follow, and if it be noneffective none will result.

RESPONSE OF ORGANISM TO STIMULI

It is a commonplace of psychology that a large part of the activity of human beings is exhibited in word *substitutions,* which is equivalent to saying that a large part of human activity is talking and thinking. Talking and thinking are primarily activities that take the place of, or are substituted for, elaborate groups of activity.[1] They are not only substitutes, but in the vast majority of cases they are condensed substitutes. The word "fire" for the child represents many months of complicated learning and behavior. The word "fear" for the adult is substituted for a group of responses of many years' duration. The description of a football game occupies from a few lines to a column in a newspaper—hours are required for the actual enactment of the game. It is, indeed, difficult to find examples of vocalization or talk which are not examples of behavior shorthand. If we stimulate a human organism by any one of a thousand effective stimuli, that individual responds with his only possible response to that stimulus. Assume now that his response is to be talk or vocalization. This word response must represent either (1) the total responses or reactivity of the organism, (2) less than the total response, or (3) apparently more than the response. (I say "apparently" because in actual human contacts undoubtedly we may appear to get from speech more than the *truth* of the situation,[2] although certainly with

1. I am avoiding the use of the term symbolizing activity, because the word symbol is so thoroughly conditioned in psychologic thinking to mean Freudian symbols. As a matter of fact, *all* words are *symbols* for objects (I include biologic objects) and their behavior toward each other.

2. By "truth" I mean truth for the particular organism. I am assuming that all human behavior, including talking and thinking, represents biologic phenomena or facts, and, as has been emphasized by Adolf Meyer so consistently, is to be viewed as of the same category as any other facts or phenomena of science. This must obviously include the meaning of words in words. This is not the place for a discussion of the philosophic meaning of "meaning." By "truth" I mean only common sense use of the word, and, by and large, *total response* could be substituted for "truth."

any kind of scientific rigor we cannot believe in more than a true response. We cannot believe that any response represents anything more than the organism plus what we put into it—i. e., the stimulus.)

Now, if the verbal response represents all of the activity of the organism to the given stimulus, we learn from vocalization the truth at this particular moment. If, on the other hand, vocalization or talk represents less than the true or total response, we must be prepared to find the organism reacting the remainder of the response in other modes of expression. This remainder is our present point of interest. In addition to talk or vocalization, we have a vast number of other expressive movements or motor responses of various gradations of complexity. Some are of a highly organized variety (movements controlled by the pyramidal tract and cortex), while others are much more diffusely and automatically organized and have for their mechanism extrapyramidal, sympathetic and visceral pathways. Under the more automatic activities, we can include changes in respiration, blushing, sweating, pupillary changes and changes in body tonus. The general body tensions manifest themselves in posture, changes in tension of a particular part of the body, general muscle relaxation or rigidity, positions of ease or discomfort. The more highly organized movements include the whole range of motor coordination known as gesture; the less organized, shifts in position, motor quiet or restlessness. The facial expression probably is a mixture of both types; certainly, part of it is of the highly organized group of activities, while part of it is as automatic as visceral activity and partakes of the general body tensions or placidity. In our ordinary study of every-day human behavior, we are apt to regard these activities as having only "casual" significance, and to slip into the attitude of believing that activity may be really casual or random. If, however, we adhere to our postulate, we see in every bit of behavior meaning and significance, though admitting that this significance and meaning may be of varying degree of pragmatic importance. In the third alternative mentioned, we assumed that it was possible for the organism apparently to exhibit in talk more than the true response to the situation or stimulus. But this is because our observation takes into account only this one mode of expression, or that we are not observing the response long enough to get the whole response. A later example, I hope, will make this clear.

Let us now consider a simple illustration of what is meant. A well-bred man is sitting at complete rest in his study just after reading a satisfactory book. His servant asks in his usual tone: "Shall I call you at your accustomed hour?" and the man responds "Yes." This

"yes" will constitute the total response of the organism to the stimulus, provided this stimulus and situation are as simple as I have assumed. The response here is entirely in one mode of expression—vocalization—and the organism has completely reacted to its stimulus. Let us suppose, on the other hand, that the man has been interrupted in an important train of thinking. The chances are that the response will be the same well-bred "yes." The stimulus has been the same as in the preceding case, but the physiologic state of the organism (the man) is different, and the total response will be different, if his "yes" is his total vocal response. The "yes" will be accompanied by activities that represent what his well-bred answer will have failed to reveal. If you ask his servant, granting that he be observant, how he knew he had made a mistake in asking the question, or had struck a bad time, he will describe some behavior which may be so slight that the casual observer would not see it. (I am assuming in this case that the "yes" is in all respects similar to the first "yes.") The evidence of irritation in the man will be expressed later through the more rapid turning of the page, or a shift of position in his chair, a general tightening of his musculature, a frown, or what not. The point is that these small bits of activity are definitely expressive of what the verbal mode of response failed to convey. We, therefore, have an example of vocalization being less than the total response or truth for the organism under that given stimulus. Suppose we assume finally that the man has made up his mind to sleep later the following morning, but in his preoccupation answers his servant automatically "yes," and before he has time to change his answer his man has disappeared. Since this response is more than the truth (the true response would have been "no"), the man will have to react out the remainder of the response to this stimulus by additional movements: for example, pushing the bell to recall the servant. If we complicate the situation and suppose the man to be concealing from his household the fact that he will not be at home the following morning, we may still find him saying "yes," but this "yes" is accompanied by a slight shortness of breath, or blushing, or restlessness in his position. His servant might tell you that he had a hunch that there was something wrong, even though not able to formulate the source of his conviction.

Let us now consider the observations of somewhat more complicated activity. A few evenings ago, while presenting an argument to a friend who was lying comfortably on a couch, I arrived at a point where through a certain vehemence, and I hope logic, of argument I had convinced him, although I knew that his general set or attitude to the whole group of ideas was antagonistic. There was a pause of a few seconds, and

then he began an unformulated, halting response made up of almost meaningless, but habitually used, sounds "um! hu!"; then his feet moved restlessly, and his whole body shifted its position on the couch. Ordinarily we call this playing for time. When confronted with the fact that he had moved, he was at first unaware of it, and then later accounted for the movement by saying that he had had a pain in his back. When asked how long he had had the pain, he said, "all evening," and admitted at the same time that the intensity of the pain had not increased. His so-called casual activity served the purpose of expressing response to a stimulus or situation that his vocalization was unable to take care of.

A patient was sent for observation to determine whether any mental disease lay behind his antisocial stealing and lying. He was a clever lad, who had learned that looking one straight in the eye was an essential part of convincement. Over and over again when confronted with a particular situation in which we had positive evidence that he had stolen a particular object, he invariably responded "no" to a question demanding confession. At the same time, he looked one straight in the eye with an air of complete honesty. Immediately, however, one could observe a backward pull to the head and the definite development of neck tension. (These body "tensions" are of great importance in psychopathology. Not only are the partial tensions of particular significance, but also the habitual general tensions which lead to body postures; their study gives valuable information.)

EXPRESSIVE REACTIONS

In passing from these examples of comparatively unorganized, nonvocal, expressive reactions (restlessness, tension, etc.) to a group of more highly organized coordinations, let us consider the simplest of these expressive reactions—the movements of the head as a whole. As far as I know, the world over, shaking of the head is used symbolically (substitutively) with the same meaning. A nod of the head is always used as a sign of affirmation; shaking the head to and fro universally means no. A great many people use these gestures as part of their ordinary expressive mechanism; that is to say, they are often used in place of words, as one uses a more positive tone of voice, or a longer, more emphatic sentence. On the other hand, one frequently observes a response in words which in itself is complete negation, yet at the same time the individual shakes his head as part of the response to the stimulus. This may mean then (and we must assume that the stimulus is uniformly equal) that the organism is revealing in the head-shaking an actual response to the stimulus or situation. If this response be a universal negative, then the head-shaking can serve the function of denying the truth of the vocalization.

I should point out that the total response represents not only aware but also unaware elements of behavior.[3] I have frequently been told by patients that no further memories or associations were forthcoming in response to my questions. If I avoided in my request any sense of urgency or insistence, so that the stimulus really represented a simple question, I could almost uniformly predict the actual truth or falsity of this response by observation of accessory or nonvocal activities. The patient's "no," if there were more material available, was either in a higher tone of voice, accompanied by a shake of the head "no" or by a general body restlessness. The following analysis illustrates the revelation of "unaware" facts through unaware activity.

In treating a patient who consulted me for occasional, unexpected, unintelligible anxiety attacks (palpitation, shortness of breath), I selected one situation in which an anxiety attack had occurred at the breakfast table. I had the patient repeat the story of this breakfast experience a great number of times, and I observed invariably that each time he told the story, between his description of drinking a glass of water and being brought two fried eggs, he always became restless in his chair. This was no more than a shift in position or a movement of his feet, and although he insisted that he could remember nothing further, I was convinced that this accessory activity represented a tension which the stimulus, i. e., the repetition of the story, caused with each repetition, but was not expressed or reacted out in his vocalizations. When I persisted in further description of this interval, the patient remembered that a vase on the breakfast table had been moved at this time and that he was able to see a person who was intimately connected with his sex life. Still, the fact that the anxiety did not begin at that point but only at

3. I use the terms "aware" and "unaware" to avoid using "conscious" and "unconscious." By "aware" activity, I mean that part of activity, either vocalized or unvocalized, that is part of the readily available expressive or manipulative mechanism of the organism; whereas by "unaware" I mean those mechanisms that are not readily available, and that may or may not have been previously vocalized and may or may not be vocalizable. If one translates this into the older nonbiologic psychology, one would include under the "aware" the conscious, easily recalled activities or memories, under "unaware" the forgotten activities or memories.

Watson, in a recent article (The Place of Kinaesthetic Visceral and Laryngeal Organization in Thinking, Psychol. Rev. **31**, No. 5 [Sept.] 1924), has emphasized that in children between the ages of 2 and 3, complicated motor activities can be carried out, and be recalled after an interval of months with only a meager accompanying word organization. He further points out that practically all essentially visceral activity (including emotional) is not accompanied by even an approximation in word organization.

I am convinced that by a further study of "conditioned" reflexes in animals and man we shall realize that much of what we now regard as "aware" behavior will be understood as behavior which has not, is not and cannot be put into words.

the actual bringing of the eggs led me to believe that this view of the other person could only have significance in connection with the eggs. The fried eggs definitely had an association in the patient's mind with semen. The patient, only a short time before, had rejected a sex experience with the boy sitting opposite him at breakfast, because the child had reached the period of pubescence and seminal discharge, which was distasteful to the patient. The first anxiety attack experienced by the patient occurred several months previously, when he had feared discovery in a sex experience with another lad of the same age. Undoubtedly, the view of the boy opposite would not have caused the attack had not the egg association called up the sex connection. I give this example in detail, because it not only represents the significance of so-called "casual" or accessory activity, but also shows the possibility of understanding specific visceral or emotional reactions in definite temporal relations. We can understand in this example, not only why the patient had an anxiety attack, but why he had it at that particular moment. The patient with whom I was dealing is honest and was making absolutely no attempt at concealment; he was completely unaware subjectively of his restlessness.

Unfortunately, in psychopathology and psychology we are apt to explain any particular reaction in terms of habit as if the manifold occurrence of the act did away with the need for explaining the determinants of a particular act. One would be as much justified in explaining the thousandth falling of an apple as due to habit. The forces causing the thousandth drop is as much the result of adequate forces as the first. The same kind of forces and adequate etiology must be looked for in every particular act or reaction. Habit can only explain the arising of new (short-cut) pathways of motor discharge. It does not do away with the need for explanation of the new pathways.

Let us now present the observations of somewhat more complicated activity. In shaking of the head, we had examples of the simplest kind of substitutive (symbolic) activities which are used constantly to enforce or negate vocalizations. The more complicated of these more highly organized, nonvocalized, expressive activities (i. e., the voluntary or pyramidal track mediated motor coordinations) are known as gesture. The gestures and accessory movements of the average person are so habitual and so peculiarly individually organized that one can usually recognize readily the person from his gesture and movements. When the vocalizations fail to complete the response the person resorts to another mode of motor expression, but from all of the available motor pathways of discharge, he makes use of just this small group of his habitual and peculiarly individual movements or gestures. It should follow that one should be able to understand the meaning of these accessory movements in relatively few patterns, or at least in a number

far less than is ordinarily thought. It is true that when one observes the behavior of some person for any length of time, one begins to see these recurrent patterns with definite meaning. For example, a man who lectures but who has no facility for words, will be observed to repeat the same gesture over and over every time a word fails. I find myself, each time I have finished dictating a sentence that seems to me satisfactory, filling in the space between my next sentence and the stenographic transcription by putting my cigaret in my mouth and brushing my trouser as if to brush away ashes. This I have observed to be true only if the sentence is satisfactory. This gesture, therefore seems to represent my own response to the satisfaction stimulus which my organism received. Again, I have observed that when a silent vocalization of my own is difficult—that is to say, when I am thinking over an idea and cannot formulate it—I find myself becoming aware of an itching on my head, and promptly make scratching movements with my hand. No doubt, my secretary recognizes my difficulty.

Bearing in mind that, as in the infant, a great deal of manual and manipulative activity is not accompanied by vocal organization (talk and thinking), and that usual, visceral (emotional) activity is seldom accompanied by words or thinking, we must expect to find a great many of the expressive responses of the individual to situational stimuli expressed in activities that are nonvocalized. In fact, one would expect to observe the nonvocalized manipulative and visceral habits, especially the unaware ones, expressed in accessory activities rather than in vocalization. To assume that these unaware ones lie in an unconscious, as forgotten word patterns, is to start with the false assumption that they were originally in vocalized patterns, i. e., that the memories had been in words. We must therefore be prepared to see the organism react out unvocalized patterns in other than vocalized modes of expression.

In ordinary manic excitement, one observes a great deal of activity that is not vocalized. The vocalization, on the other hand, is constantly giving out meaning which on the face of it is untrue—that is to say, one constantly hears all sorts of flighty delusional material. According to my interpretation, the other activities accompanying these false vocalizations serve a function of belying or contradicting the talk of the patient. As a corollary to this, one could expect that, if the accessory movements were eliminated, the total response could be forced out through vocalizing channels, and the more completely one could direct the activity into talk, the more nearly would it represent the total response of the organism—and the more nearly it represented the total response of the organism, the more nearly would it represent the truth for that organism in its particular physiologic state at that moment. On repeated occasions. I have observed recovering manic patients, who could be forced into giving up accessory movements; I could immobilize the body

activities by requesting the patient to stop moving. One immediatelv observes that the talk of the patient becomes much more rational. The last of the expressive activities that one can force the patient to abandon. are the facial movements—frowning, smiling, etc. Smiling in many cases plays the rôle of putting a double meaning on what is said, i. e., it serves to deny the real meaning of one's words (I do not mean to say that this is the only meaning of smiling). I have frequently observed that if I ask the patient not to smile after having immobilized his body, I begin to get talk, having a far greater approach to truth than I have previously obtained. A few days ago, while making the rounds of the ward, I tried the experiment on a patient, and the intern, who knows nothing of these notions, said: "This is the straightest account we have yet obtained from the patient." It is true that the general physiologic state of the organism in a manic patient is much increased over his customary state, and that to get a response which we should call normal, one has to use minimally effective stimuli to counterbalance the patient's excitability. Actually, all treatment is aimed in this direction. We remove patients from exciting situations, minimize stimuli by putting them in warm baths, etc. In the case cited, we were careful to talk simply to the patient, in a tone of voice just loud enough to be heard, so that the stimulus would be barely effective.

CONCLUSIONS

The method of approach outlined seems to me to serve several distinct functions: First, it presents an objective method of observation which takes a whole group of activities out of the so-called casual or random activities and sees in them material for scientific study; second, it has led to valuable aid in the study of psychopathology; third, it attempts to see human psychology in terms of biologic stimulus and response under a rigorous system of causal sequences.

It has been urged by my colleagues that even if the view be correct, the method is valid only if one uses an intuitive method. They are correct in recognizing the great need for exercising caution in interpreting results, and that only skilful observation will bring results. I admit that the method is intuitive, provided it be clear that by intuitive they mean what I do. I mean a capacity to mobilize one's own past experience of behavior without going through all the logical formulations necessary to convince the hard-headed or sceptical bystander. In this method, I see nothing of the mystical character usually associated with the word intuitive. When we begin to understand the real automaticity of human behavior—including thinking—and drop the obviously false notion that all thinking is purely voluntary and purposive, we shall not be astounded by this kind of observation, nor shy at seeing meaning in casual or random activity.

MOTOR DISTURBANCES IN PERIPHERAL NERVE LESIONS

(A) MUSCLES INVOLVED IN PARTIAL LESIONS (B) ORDER OF RESTORED MOTION *

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The order in which movement is restored to muscles paralyzed as the result of peripheral nerve lesions and the particular muscles involved in partial or dissociated nerve lesions have a sufficient constancy to attribute to each nerve a clinical individuality (Marie and Benisty [1]).

This analysis was made from a certain number of records which were competent, selected from a large amount of material. They may be divided into three groups: first, records of cases seen in France soon after injury; second, records of cases seen in U. S. General Hospital No. 28 a number of months after injury, and third, an analysis of motor disturbances in median nerve lesions from the whole material in the Surgeon-General's office.

The records of the group studied at U. S. General Hospital No. 28 are especially adapted for this study, as muscle power was ascertained by dynamometric examination, described elsewhere (Pollock [2]).

ULNAR NERVE

The level of the lesion at times is the determining factor of a lesion which does not produce paralysis of all the muscles supplied by the ulnar nerve. When the lesion affects the nerve distal to a point 1 inch below the internal condyle of the humerus, the flexor carpi ulnaris is spared.

Benisty [3] states that the most common type of partial paralysis is that produced by a wound in the axilla or arm, with paralysis of the interossei and hypothenar muscles, and merely paresis of the flexor profundus and flexor carpi ulnaris.

The intraneural localization of fibers destined to supply certain muscles, as pointed out by Stoffel, Marie, Meige, Gosset, Déjerine and Mouzon and Kraus, is given by Benisty as an explanation of the constancy of certain partial lesions of various nerves. However, it may be well to remember that Dustin believes that he has conclusively shown that between each branch of a nerve there is a reassembling of nerve fibers so that a long intraneural pathway does not exist.

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* Read before the Chicago Neurological Society, Jan. 15, 1925.

1. Marie, Pierre, and Benisty, Mme. A.: Rev. neurol., 1915.
2. Pollock, Lewis J.: Surg., Gyenc. & Obst. **38**:223-228 (March) 1924.
3. Benisty, Mme. A.: The Clinical Forms of Nerve Lesions, Mil. M. Manuals, Univ. of London Press, 1918.

In the cases observed soon after injury, of eleven in which the injury occurred in the arm, all muscles were weak only in five cases; the hypothenar were paralyzed in four cases, the interossei in two cases, and all muscles were paralyzed in one case. In seventeen cases in which the injury was in the forearm, all muscles were weak in six cases, the hypothenar group was paralyzed in four, the interossei in three, all muscles were paralyzed in three cases, and the adductor of the thumb and the flexor of the wrist in one each (Fig. 1, *a* and *b*).

It may be seen, then, that whether we are dealing with a lesion in the arm or in the forearm, the same order of frequency of paralyzed muscles exists. Those paralyzed most frequently were, as noted by Benisty, the hypothenar group and the interossei.

The order of recovery in ulnar nerve lesions is given by Benisty as flexor carpi ulnaris, flexor profundus digitorum, and then the small hand muscles which recover very slowly. The Committee on Injuries of the Nervous System of the Medical Research Council [4] stated that a better functional recovery is to be expected in the flexor carpi ulnaris, flexor profundus digitorum and in the abductor minimi digiti than in the other muscles.

It was pointed out before that, because of supplementary motility, the strength of movement of the phalanges was a misleading sign of recovery. Because of this, the cases seen some months after injury were analyzed from the standpoint of residual paralysis. Of twelve recovering cases of arm injury, the hypothenar group was paralyzed in eight cases, the interossei in four, the flexor carpi ulnaris in two, the adductor of the thumb in two, and all muscles in one. In fourteen cases of injury in the forearm, the interossei were paralyzed in twelve cases, the hypothenar in nine, the adductor of the thumb in four, and all muscles were weak in one (Fig. 1, *c* and *d*).

In another series, it was noted that of twenty-one cases in which movement had returned in the small muscles of the hand, the adductor of the thumb had recovered in four cases and the abductor of the little finger in three cases.

This is in general agreement with the authors quoted, in that recovery occurred most regularly in the flexor carpi ulnaris and late in the small muscles of the hand, but the adductor of the thumb recovered more frequently than the abductor of the little finger.

MEDIAN NERVE

As in the ulnar, the level of the lesions determines the cases with paralysis of only some of the muscles supplied by the median nerve. No muscular branches are given off in the arm. The branches to the

4. Medical Research Council: The Diagnosis and Treatment of Peripheral Nerve Injuries, London, 1920.

superficial flexors of the middle, ring and little fingers, the pronator radii teres and the flexor carpi radialis are given off in the upper part of the forearm, whereas the superficial flexor of the index finger is given off the median in the lower third of the forearm.

Partial Lesions.—Benisty states that the flexion of the index finger is the most defective movement, though opposition of the thumb is very poor as well. There is frequently imperfect flexion of the middle finger and of the second phalanx of the thumb. The pronators and flexors of the wrist are either not affected at all or slightly paretic.

Of sixteen cases seen in U. S. General Hospital No. 28 after injury in the arm, the opponens was paralyzed in eight cases, all were weak in seven cases, the flexor of the thumb in four cases, the flexor of the index finger in three cases and the palmaris longus in one case. Of eleven cases of injury in the forearm, the flexor of the index finger was paralyzed in five cases; all muscles were weak in four cases; the

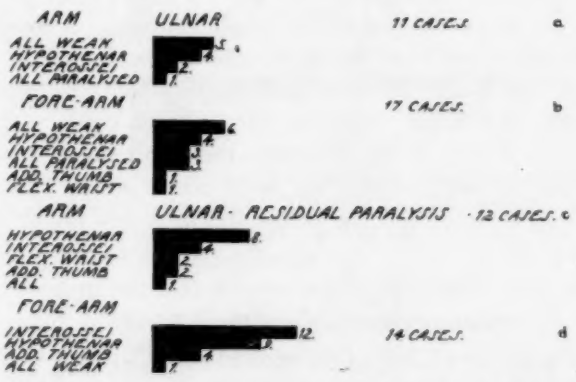

Fig. 1.—*a* (eleven cases) and *b* (seventeen cases), muscles involved in lesions of ulnar nerve in arm and forearm, respectively; *c* (twelve cases) and *d* (fourteen cases), residual paralysis in lesions of ulnar nerve in arm and forearm, respectively.

flexor of the thumb, the abductor of the thumb and the opponens were paralyzed in two cases each, and the palmaris longus in one case. As in Benisty's material, when dissociated paralysis occurred, the flexor of the wrist was rarely affected. However, the opponens seemingly was more severely and more frequently affected than the flexor of the index finger in lesions of the arm. Further reference to this will be made under residual paralysis. In lesions of the forearm, the flexor of the index finger was more frequently involved.

In an analysis of the records in the Surgeon-General's office representing all of the American material, of fifty-nine cases of partial lesions in injuries of the arm, all muscles were weak in thirty-three cases, small muscles of the hand were paralyzed in nine cases, all muscles

in eight cases, the flexor of the index finger in seven cases, the flexor of the thumb in three cases, the abductor of the thumb in two cases, the opponens in two cases, and the palmaris longus in one case (Fig. 2, *c*).

Of fifty-six cases of injury in the forearm, the small muscles of the hand were paralyzed in thirty-one cases, all were weak in fifteen cases, all were paralyzed in five cases, the flexor of the index finger in five cases, the flexor of the thumb in two cases and the abductor of the thumb in one case. It will be noted that the small muscles of the hand are most frequently paralyzed, and next to them the flexor of the index finger. In lesions in the forearm, paralysis in the small muscles of the hand is more frequent, because the lesion is below the point where the fibers to the flexors of the fingers are given off (Fig. 2, *d*).

Of twenty-five cases of partial lesions which were sufficiently severe to warrant operation in arm injury, all were paralyzed in nine cases, all were weak in seven cases, the small muscles of the hand were paralyzed in six cases, the flexor of the index finger in five cases, the flexor of the thumb in two cases, the abductor of the thumb in two cases and the flexor carpi radialis in one case. Of seventeen similar cases of injury in the forearm, the small muscles of the hand were paralyzed in seven cases, all were weak in six cases, all were paralyzed in four cases, and the flexor of the index finger in one case (Fig. 2, *a* and *b*).

It will be noted that whether severe or not, the order of frequency of paralysis of certain muscles is the same.

Recovery.—Benisty thinks that recovery of function of the muscles in lesions of the median nerve takes place in complete and partial paralysis in very much the same way. The pronator and palmar muscles regain their functional activity first, then the flexors of the middle finger and afterward the flexor of the thumb. Flexion of the index finger and opposition of the thumb are impaired for a very long time.

Stopford's [5] statistics show that in fourteen cases of suture of the median nerve in the lower third of the forearm, the abductor brevis pollicis recovered first in five cases; of ten cases at the bend of the elbow above the motor branches, in one case the pronator and flexor carpi radialis recovered first, and in three cases the abductor brevis pollicis; in eight cases of suture in the arm, the pronator, flexor carpi radialis and palmaris longus recovered in one case and the abductor brevis pollicis in three cases.

From the Surgeon-General's material of twelve cases of injury in the arm and spontaneous recovery, there was return of function in

5. Stopford, quoted by Medical Research Council, Footnote 4.

the flexor of the index finger in seven cases, in the flexor carpi radialis in five cases, in the flexor of the thumb in five cases and in the small muscles of the hand in one case. Of nine cases of injury in the forearm, there was return of function in the flexor of the index finger in five cases, in the flexor carpi radialis in two cases, and in the small muscles of the hand and abductor of the thumb in one each. Following suture

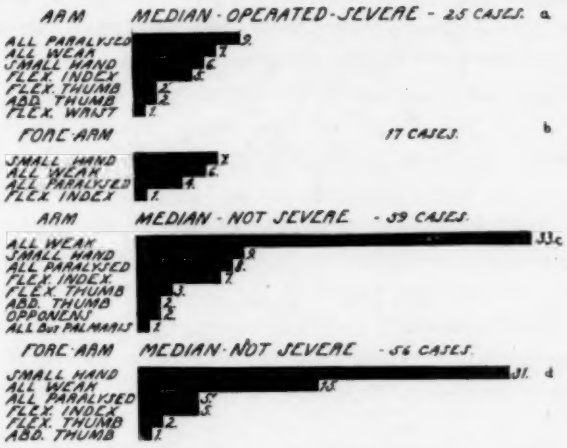

Fig. 2.—*a* (twenty-five cases) and *b* (seventeen cases), muscles involved in severe but partial lesions of the median nerve in arm and forearm, respectively; *c* (fifty-nine cases) and *d* (fifty-six cases), muscles involved in partial lesions of median nerve in arm and forearm, respectively.

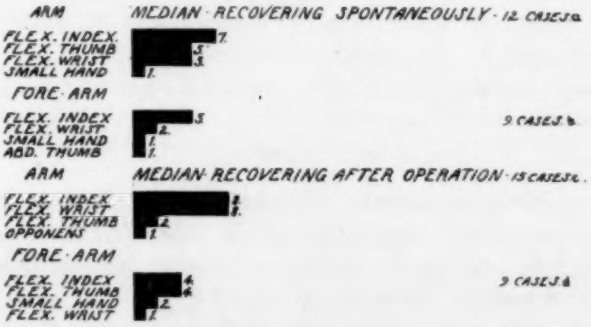

Fig. 3.—*a* (twelve cases) and *b* (nine cases), muscles recovering spontaneously in lesions of the median nerve in arm and forearm, respectively; *c* (fifteen cases) and *d* (nine cases), muscles recovering after suture of the median nerve in arm and forearm, respectively.

in the arm in fifteen cases, there was a return of function in the index finger in eight cases, in the flexor carpi radialis in eight cases, in the flexor of the thumb in two cases and in the opponens in one case. Of nine patients sutured in the forearm, there was a return of function

in the flexor of the index finger in four cases, in the flexor of the thumb in four cases, in the small muscles of the hand in two cases, and in the flexor of the wrist in one case (Fig. 3, *a*, *b*, *c* and *d*).

The large number of cases in which there was a return of function in the flexor of the index finger may in part be due to an error because of misinterpretation of supplementary motility. Ignoring this muscle, it may be seen in these cases that the flexors of the wrist and the thumb recover first. This differs from Stopford's figures in the few cases of recovery of the abductor of the thumb.

Further light may be thrown on the problem in a study of residual paralysis.

Of eighteen cases of injury in the arm, residual paralysis remained in the flexor of the index finger in eleven cases and in the small muscles of the hand in twelve cases. Of twelve cases in the forearm, residual paralysis remained in the small muscles of the hand in eight cases and in the flexor of the index finger in six cases. It is evident that even if recovery does *begin* early in the flexor of the index finger, it is very late in its completion. When recovery occurs in the small muscles of the hand, the opponens is the last to recover.

ULNAR AND MEDIAN NERVES

Partial lesions of the ulnar and median nerves produce many interesting types of dissociated paralysis, but because of widespread supplementary motility recovery of function is difficult to study and a definite pattern of involvement cannot be determined. It may be said that in incomplete lesions of either ulnar or median nerves, weak movements of the phalanges of the finger, if interpreted alone, are insufficient evidence as to whether one of these nerves is severed, and as to which one may be severed.

MUSCULOSPIRAL

Owing to the fact that the branches to the supinator longus, and less frequently the extensor carpi radialis, arise from the nerve above the middle of the arm, these muscles may escape harm in case of injury in the lower fourth of the arm.

Of fourteen cases of injury in the arm observed in France, the extensors of the thumb were paralyzed in eight cases, of the wrist in seven cases, of the fingers in four cases; all muscles were weak in three cases; all were paralyzed in two cases, and the supinator was paralyzed in one case. Of ten cases of injury in the forearm, all muscles were weak in four cases; the extensors of the fingers were paralyzed in four cases, the extensors of the thumb in two cases, and all were paralyzed in one case (Fig. 4, *a* and *b*).

At times the extensors of the wrist may be paralyzed and those of the fingers unaffected; again, the extensor of the middle finger may be spared. The relatively small number of cases showing paralysis of the extensors of the fingers may be due to misinterpretation of supplementary motility.

Recovery.—According to Oppenheim, the muscles recover in direct relation to the length of nerve fibers regenerating. This is not the case. Benisty states that the order of recovery is nearly always the same. The common extensor is restored only after the radial muscles; the middle, ring and little fingers begin to extend first and the index last; but sometimes the index and middle fingers first recover their movements. The extensors of the thumb and the abductor of the thumb regain their power last.

Of sixteen cases in which operation was performed in the arm, return of function was observed in the extensors of the wrist in ten

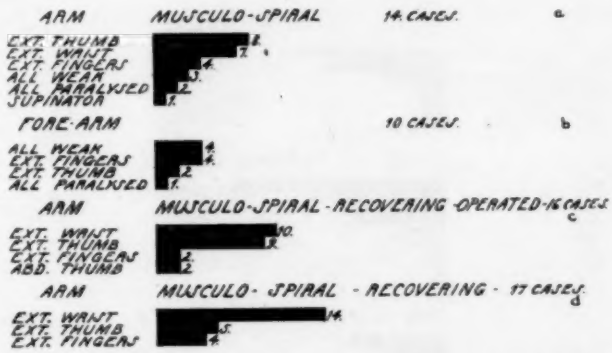

Fig. 4.—*a* (fourteen cases) and *b* (ten cases), muscles involved in partial lesions of the musculospiral nerve in arm and forearm, respectively; *c* (sixteen cases) and *d* (seventeen cases), recovery following operation of musculospiral nerve in arm and forearm, respectively.

cases, in the extensor of the thumb in nine cases and in the extensors of the fingers in two cases, in the abductor of the thumb also in two cases. Of seventeen cases in which operation was performed in the forearm, power returned in the extensor of the wrist in fourteen cases, in the extensors of the thumb in five cases and in the fingers in four cases (Fig 4, *c* and *d*).

These observations differ from those of Benisty, in that the extensors of the thumb recovered before those of the fingers. As to the order of recovery of the extensors of the fingers and thumb, those of the thumb showed some recovery first in sixteen of thirty cases, but when this did not occur the thumb was almost the last to recover. The extensor of the middle finger was one of the first to recover in seventeen of thirty cases, and the index finger was the last to extend in ten cases.

Rarely return of function was seen in the long abductor of the thumb and in the extensor of the thumb before that in the extensors of the wrists.

SCIATIC

The branches to the hamstring muscles are most frequently not included in lesions of the sciatic nerve. Dissociated or partial paralyses may be due to greater damage to either the external or internal popliteal branches, or partial injury to both. Operative and clinical experience shows that the external popliteal suffers greater damage, as a rule, than does the internal popliteal.

Of thirty-seven cases seen soon after injury, the external popliteal was paralyzed and the internal popliteal was weak in twelve cases, all muscles were weak in ten cases, the internal popliteal was paralyzed and the external popliteal was weak in four cases, the external popliteal alone was paralyzed in two cases, the internal popliteal was weak in

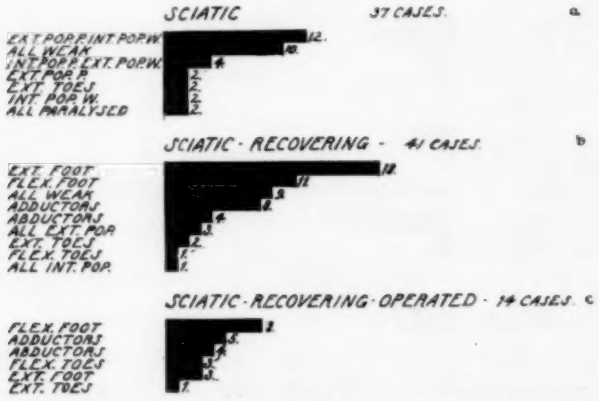

Fig. 5.—*a* (thirty-seven cases), muscles involved in partial lesions of sciatic; *b* (forty-one cases), muscles recovering in partial lesions of sciatic; *c* (fourteen cases), muscles recovering following suture of sciatic.

two cases, the extensors of the toes were weak in two cases, and all muscles were paralyzed in two cases (Fig. 5, *a*).

Recovery.—According to Benisty, the muscles which recover first are, according to the individual case, the tibialis anticus, the peroneus longus or the gastrocnemius; rarely, recovery begins in the tibialis posticus. The flexors or extensors of the toes are always the last to recover.

The Medical Research Council reported that, as a rule, the internal popliteal nerve shows signs of return of function earlier than does the external popliteal. Stopford's records of sutured cases show that the order of recovery is: gastrocnemius, tibialis anticus, extensor longus digitorum, extensor longus hallucis.

The records of the Military Orthopedic Hospital, Shepherd's Bush, gives the order of recovery in eight cases as: tibialis posticus, gastrocnemius, tibialis anticus, extensor longus digitorum, extensor longus hallucis, extensor brevis digitorum and peroneus longus.

In forty-one recovering patients, there was return of function in the tibialis anticus in eighteen cases, in the gastrocnemius in eleven cases, in all muscles in nine cases, in the tibialis posticus in eight, in the peronei in four, in all muscles of the external popliteal in three, in the flexors of the toes in one case, and in all muscles supplied by the internal popliteal in one case (Fig. 5, *b*).

In fourteen cases following suture, function returned first in the gastrocnemius in eight cases, in the tibialis posticus in five cases, in the peronei in four cases, in the flexors of the toes in three cases, in the tibialis anticus in three cases and in the extensors of the toes in one case (Fig. 5, *c*).

In severe cases and following suture, return of function in the branches of the internal popliteal occurred earlier than in those of the

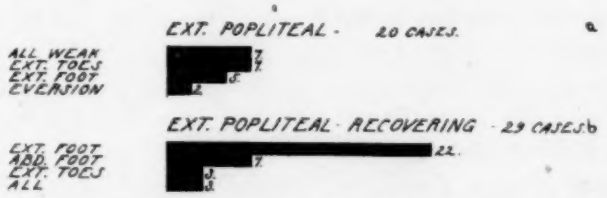

Fig. 6.—*a* (twenty cases), muscles involved in partial lesions of the external popliteal; *b* (twenty-two cases), muscles recovering in partial lesions of the external popliteal.

external popliteal; extension of the toes was very late in appearance, and in spontaneously recovering lesions, flexion of the toes occurred very late.

In this respect, our findings differ from Stopford's and those of the Medical Research Council, and agree with the findings of Benisty.

EXTERNAL POPLITEAL

Benisty gives the order of recovery as: tibialis anticus, peronei, extensor longus digitorum and extensor propius hallucis. Stopford agrees with this, whereas the records of the Military Orthopedic Hospital put the extensor longus digitorum first.

Of twenty-nine patients spontaneously recovering, the tibialis anticus recovered in twenty-two cases, the peronei in seven cases, the extensor longus digitorum in three cases and all the muscles in three cases. In six cases showing recovery out of twenty-seven cases sutured, the order was: tibialis anticus, extensor longus digitorum and peronei (Fig. 6, *a* and *b*).

SUMMARY

Ulnar Nerve.—Whether slightly or severely injured, the muscles most frequently involved in ulnar nerve lesions are the same. The hypothenar group of muscles and the interossei were most frequently affected. When the long flexors were affected, all the muscles were weak.

The order of recovery was as follows: the flexor carpi ulnaris, then the small muscles of the hand. Because of supplementary motility, accurate measurement of the flexor profundus could not be made. Of the small muscles of the hand, the adductor of the thumb recovered more frequently than the abductor of the little finger.

Median Nerve.—In conformity with other observers, it was found that the flexor carpi radialis was rarely affected. Of the small muscles of the hand, the opponens was most frequently affected. The flexor of the index finger did not seem to be injured as often as the small muscles of the hand. Whether severe or not, lesions of the median nerve produce the same order of frequency of muscles involved.

Recovery occurred first in the flexor carpi radialis and flexor longus pollicis. Movement seemed to begin to return early in the flexor of the index finger, but from a study of residual paralysis, it was evident that flexion of the index finger remained imperfect for a very long time, comparable to the opponens.

Ulnar and Median Nerves.—Combined lesions of these nerves produced many patterns of weakness. Only one important observation was made, namely, that weak movements of the phalanges of the fingers, if interpreted alone, are insufficient evidence as to whether one of these nerves is severed and as to which one may be severed.

Musculospiral Nerve.—At times, the extensors of the wrist may be paralyzed and those of the fingers be unaffected; at times, the extensor of the middle finger may be spared.

The extensors of the wrist were first to recover, then the extensors of the thumb, then of the fingers. Extension of the middle finger returned before that of the index finger.

Sciatic Nerve.—The muscles supplied less by the external popliteal branch suffered greater damage, as a rule, than those supplied by the internal popliteal.

The order of return of function was the gastrocnemius, tibialis posticus, peronei, tibialis anticus, extensor longus digitorum and then the flexors of the toes.

External Popliteal Nerve.—The order of recovery was the tibialis anticus, peronei, extensor longus digitorum, extensor proprius hallucis.

25 East Washington Street.

News and Comment

PROGRAM FOR MEETING OF ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

The following communications have been offered for the meeting of the Association for Research in Nervous and Mental Disease to be held in New York City, December, 1925.

1. Historical Survey: Pre-Kraepelinian Conceptions. Kraepelin's Contribution (dementia praecox), Later Developments. Dr. Adolf Meyer, Baltimore.
2. Criteria for the Definition and Delimitation of the Schizophrenic Reaction. Dr. C. M. Campbell, Boston.
3. Frequency of Schizophrenia in Relation to Race, Nativity, Age, Sex, and Environment. Dr. Horatio M. Pollock, Albany.
4. Hereditary and Familial Relations of Schizophrenia. Dr. Albert M. Barrett, Ann Arbor.
5. Heredity in Schizophrenia. Dr. Abraham Myerson, Boston.
6. The Outcome of Schizophrenic Reactions Observed in Soldiers During the War. Dr. Thomas W. Salmon, New York.
7. Constitutional Factors in Schizophrenia. Dr. Theophile Raphael, Ann Arbor.
8. Mental and Emotional Components of the Personality in Schizophrenia. Dr. George S. Amsden, Albany.
9. Affective Experience in Early Schizophrenia. Dr. Harry S. Sullivan, Baltimore.
10. Emotional Maladjustment and Possibilities of Readjustment in Schizophrenia (The Influence of Jealousy in Schizophrenia). Dr. Edward J. Kempf, New York.
11. Psychogalvanic and Association Studies in Schizophrenia and Kindred Conditions. Drs. Richter and Syz, Baltimore.
12. Brain Changes in Schizophrenia. Dr. Charles B. Dunlap, New York.
13. Lipoid Degeneration Products in the Thalamus and Globus Pallidum of Schizophrenia. Dr. Walter Freeman, Washington.
14. Histological Study of the Endocrine Glands in Schizophrenia. Dr. Bertram D. Lewin, New York.
15. Calorimetry of Schizophrenic Blood Vessels. Dr. Nolan D. C. Lewis, Washington.
16. Biology of Sex in Schizophrenia. Dr. Charles E. Gibbs, New York.
17. Prognosis of Schizophrenia. Dr. Edward A. Strecker, Philadelphia.
18. Course and Outcome of Schizophrenia. Dr. Eleanora B. Saunders, Baltimore.
19. Deterioration in Schizophrenia: Its Occurrence, Characteristics and Nature. Dr. George H. Kirby, New York.
20. Precipitating Causes of Schizophrenia. Dr. R. H. Hutchings, Utica.
21. Alcoholism and Schizophrenia. Dr. M. S. Gregory, New York.
22. Schizophrenic Reactions in Prisoners. Dr. Herman M. Adler, Chicago.
23. Schizophrenic Reactions and the Psychoneuroses. Dr. A. A. Brill, New York.

24. The Language of Schizophrenia. Dr. William A. White, Washington.
25. Endocrine and Biochemical Studies in Schizophrenia. Dr. Karl M. Bowman, Boston.
26. The Weight of the Heart in Schizophrenia and Other Mental Diseases. Dr. Marjorie Fulstow, Boston.
27. Treatment of Schizophrenia. Dr. H. A. Cotton, Trenton.
28. Schizophrenia and Epidemic Encephalitis, Their Alliances and Differences. Dr. Smith Ely Jelliffe, New York.
29. Gastro-Intestinal Motor Functions in Schizophrenia. Dr. George W. Henry, White Plains.
30. Episodic or Transitory Schizophrenic Reactions. Dr. Sidney I. Schwab, St. Louis.

Abstracts from Current Literature

THE PRESENT STATUS IN THE TREATMENT OF METASYPHILIS. W. WEYGANDT, Ztschr. f. d. ges. Neurol. u. Psychiat. **96**:7 (April) 1925.

Möbius about three decades ago spoke of tabes and general paralysis as metasyphilitic infections, and while he looked on the process as syphilitic, he thought that the effect probably was produced by some toxin which attacked the nervous system under certain favorable conditions. The demonstration of spirochetes by Noguchi proved the syphilitic nature of these conditions. Virchow, in 1898, however, stated that definite anatomic proof of syphilitic infections in tabes and paresis is seldom found, and the inefficiency of antisyphilitic treatment in these diseases was further demonstration of their nonsyphilitic nature.

Several theories have been brought forward to explain the occurrence of tabes and paresis: 1. One theory is that of hereditary disposition, which bases its argument on the difference in the occurrence of metasyphilitic conditions in civilized and barbarous or primitive nations. Cases of tabes and paresis, however, occur among uncivilized peoples. 2. Kraepelin propounded the theory that during the last hundred years the human constitution has changed so as to make it susceptible to the toxin which causes these metasyphilitic conditions. Proof of this is found in the increased number of cases of paresis and tabes during the last century, and their infrequency before the nineteenth century. 3. Erb advanced the conception of definite neurotropic strains of spyrochetes with a predilection for attacking the central nervous system. Plaut and Mulzer corroborated this experimentally by finding a neurotropic strain in a dog, after several hundred transfers. Furthermore, Mulzer and Plaut induced spinal fluid changes in a dog by the injection of paretic cortex. At necropsy the brain showed pictures similar to that seen in human paresis. Levaditi claims to have isolated four neurotropic strains of spirochetes. O. Fischer would differentiate a tropic strain for paresis, tabes and cerebrospinal syphilis, but Weygandt does not subscribe to this idea. The question is complicated, moreover, by the fact that there are intermediary stages between tabes and paresis just as there are between paresis and cerebral syphilis. Weygandt believes that the conception of neurotropic strains, despite the work of Plaut and of Levaditi, needs confirmation. 4. Still another idea as to the origin of tabes and paresis has been advanced by Nonne, who considers these conditions as artificially produced by improper treatment. Statistics by Krou and Pilcz show that the shorter the treatment the shorter the period between the primary infection and the occurrence of tabes and paresis. Gärtner has reported a marked increase in paresis in recent years in Germany. In 178 prostitutes with positive fluid findings Kyrle found that 33 per cent. were untreated, 22 per cent. were treated with mercury, and 44 per cent. were treated insufficiently with mercury and arsenic. Experimental proof of the fact that insufficient treatment can cause these conditions in the nervous tissue is furnished by Plaut, who infected a dog with a non-neurotropic strain of spirochetes, and then by insufficient treatment with arsphenamin induced positive spinal fluid findings. Weygandt himself believes that a great many patients are insufficiently treated, and that more intensive treatment will yield better results. He believes the spirochetal infection causes the production of immune bodies, and that the occurrence of skin lesions indicates the index of protectives for the

internal organs, especially the nervous system. The fluid becomes infected in the secondary stages, and the spirochetes remain dormant for some time in the meninges and blood vessels only to become active later and disseminate in the nervous tissue. Lewandowsky has demonstrated that tubercle bacilli may become activated in this way. 5. Gennerich, too, has propounded a theory regarding the pathogenesis of general paresis. Briefly, his theory is that as a result of inadequate treatment the virulence of the syphilitic infection is mitigated and the body defence weakened. Hence a chronic latent syphilitic meningitis arises and produces adhesions of the pia to the cortex. In proportion to the amount of this adhesion and degeneration, there is a more or less abundant invasion of the cortical parenchyma by the cerebrospinal fluid, for one of the functions of the pia is to prevent the brain tissue from being soaked with fluid. The invasion of the cortex by the fluid causes the paretic condition. Weygandt does not believe that hypothesis has been amply proved. 6. Hauptmann believes that the hyperpermeability of the meningeal vessels permits toxic substances to enter the fluid from the blood, in this way causing infection. 7. Still others believe in a proteolytic process.

The treatment of tabes and general paresis goes far back. The middle generation of neurologists and psychiatrists are descendants of the period of therapeutic nihilism characterized by the attitude of Möbius that there is no cure for paresis. Weygandt delves far into the past and produces evidences of cures of psychoses from Galen and Hippocrates. Nasse, in 1870, reported improvement in paresis by malaria and also variola. Hemming, in 1877, described a seven year remission in paresis after typhoid. Cases of improvement and apparent cure in paresis have been reported after measles, erysipelas, variola, pneumonia, diphtheria, cholera, phlegmons, abscesses and even chorea. Weygandt reports a patient of his who had a remission of one and a half years after a phlegmon, and calls attention to remissions after influenza and typhoid fever. The various other methods of treatment are discussed, e. g., mercury, endolumbar treatments and other methods. Gennerich reports very good results by means of the endolumbar method, but Weygandt has seen no results in his paretic patients. Knauer's method of arsenic injection into the carotid has produced no marked results. Cistern treatments as advocated by Purves-Stewart and Nonne with good results have been of doubtful efficacy in the hands of the author. The same is true of cerebral injections. Numerous methods are detailed, but with no striking results. Silver arsphenamin seems to be of some value in decreasing the serologic findings. Newer arsenical and bismuth preparations are mentioned. Unspecific remedies have recently been used with some success. Besides substances such as albumose, deutero-albumose, casein and milk preparations, other substances such as "nucleinate" preparations have been used. Donath has reported 47.6 per cent. good remissions and 24 per cent. slight remissions in twenty-one patients treated with sodium nucleinate. Tuberculin too has been used since 1900 as a means of treatment in metasyphilis. Weygandt reports remissions in one third of his cases, much more than in spontaneous remissions. Wagner-Jauregg has reported twenty-six per cent. of remissions in paretic persons treated with polyvalent staphylococcus vaccine, and since 1917 Wagner-Jauregg has used malaria for the treatment of paresis, with startling results.

Weygandt points out that the use of malaria in the treatment of paresis is attended with certain dangers. Malaria itself is likely to affect the nervous system; in the severe tropical forms one may have epileptiform convulsions, unconsciousness, spasms and local twitchings, delirium and coma. In chronic

malaria one may have all kinds of neuralgia and neuritides, optic neuritis, ocular palsies and often a polyneuritis. Besides, deaths which follow immediately after or during the malaria are not uncommon. The danger of infection by contact is very real, and Weygandt states emphatically that this treatment should be confined to the hospital and not tried in private practice. Following the "cure" there often occurs an anemia of more or less severity. In one of the author's cases a pernicious anemia occurred more than six months after the malaria cure. Among several hundred cases of paresis treated with malaria by Weygandt there occurred complete remissions in 26 per cent. and satisfactory remissions in 18 per cent, the remissions being more complete and longer than spontaneous remissions. In many cases there occurred an improvement in the reaction to light, and disappearance of the stiff pupil occurs, even though not common. The patellar reflexes often return and improvement in sight with optic atrophy often occurs. The serologic improvement is even better than the clinical. The pleocytosis disappears, the globulin reaction becomes weaker, and the blood Wassermann test becomes negative, usually before the fluid Wassermann test. Wagner-Jauregg considers the fluid reactions unimportant for prognostic significance. Weygandt reports on the histologic examination of brains of paretic persons treated by malaria. In general, he says that there is a freedom from spirochetes and a tendency of the inflammatory phenomena to disappear, sometimes completely, just as in stationary paresis. Treatment of paresis by recurrent fever, however, has yielded satisfactory results with a fairly high mortality.

ALPERS, Philadelphia.

GERLIER'S DISEASE. KARL REHSTEINER, Schweiz. med. Wchnschr. **55**:410 (May 7) 1925.

In 1887, Gerlier reported the occurrence of a new syndrome "vertige paralysant," noted in patients of his district. The syndrome was characterized by attacks of vertigo, associated with ptosis of the eyelids and palsies of the voluntary muscles, especially those of the neck. Hatenhoff, Pégaitaz and David had also noted epidemics of this condition in the same region. In 1888, two Japanese physicians reported a similar condition in a province in Japan. With the exception of these two areas, no cases have been reported in the literature.

In 1889 Gerlier published a monograph on this subject, and named these characteristics of the condition: muscular palsies, visual disturbances and dizziness, pains in the neck and back. The palsies affected only voluntary muscles, especially extensor groups. The levator palpebrae superioris were most commonly involved. Next in frequency were the neck muscles producing a falling forward of the head on the chest (Kopfhängen). The extensors of the limbs were frequently involved and occasionally to such an extent as to produce inability to use them. The muscles of mastication and facial muscles were rarely affected. Visual disturbances and vertigo accompanied or preceded the muscular weakness. The dizziness, which might be so severe as to cause the individual to fall, was not associated with illness or loss of consciousness. The visual disturbances were usually in the nature of blurring or dimness. Diplopias were rare. No pupillary changes were noted. In a few instances a slight hyperemia of the fundi was noted.

The neck and back pains were variable, at times severe and again entirely absent. The attacks were described as usually of short duration, a single attack lasting but a few minutes. However, they might occur in rapid succession extending over several hours and representing a type of crisis.

Gerlier distinguished three types of attacks which differed from one another in intensity: (1) ptosis and mild visual disturbance (type de l'endormi), (2) ptosis and cervical weakness so that head would fall forward, (3) dizziness, weakness of the limbs and a reeling gait.

Prodromes were rather rare, although occasionally a feeling of hunger or salivation preceded the attack. The condition would usually begin during the summer months. The attacks would gradually become more frequent, increasing in intensity so that from ten to twenty might occur in one day. There would then be a slow improvement, so that with the onset of cold weather the attacks would disappear. Attacks were rarely noted in winter. While a recurrence of the condition was uncommon, the Japanese writer reported recurrences in certain persons for a period of twenty years. In no case did death result during an attack.

Between the attacks the patients were usually quite well. Occasionally a slight ptosis or general muscular weakness would exhibit itself. In all instances, it was noted that the affection occurred in people who worked about the same barn; members of the same families who did not work about the barn were not affected. Gerlier believed that the virus was probably produced in the manure. People working in dark, poorly ventilated barns or who had to care for the manure were affected. In northern Japan, where the barns and houses were under the same roof, all members of the family were affected. Cases have also been reported of direct transmission to nurslings through the mother's milk. In 1914 Couchoud, who studied the condition in Japan, is said to have found a staphylococcus-like organism in the human milk and in the spinal fluid of affected patients. Cultures injected into cats produced a transient picture identical to various syndromes. Gerlier had previously noted that cats, chickens, horses and other animals were affected by the disease. The spinal fluid of infected cats also showed cocci and again produced the disease in other experimental animals.

Since 1900, no cases of Gerlier's disease have been reported in Europe. In October, 1924, the author observed four cases, the patients constituting an entire family who lived together in the same house without servants. Investigation of its locality brought to light another family of four members in which all the males were affected, the mother being free. Of these seven patients, the writer examined five. In the first family (L) the father first showed signs of trouble toward the end of July, 1924. The first attacks were mild and of short duration, consisting of ptosis and blurring of vision. Later the attacks became more severe so that he could not raise the lids, and occasionally diplopia and weakness of the muscles of the neck occurred. There were no pains, and the remaining musculature was not affected except that during the attack he felt a general weakness. In the more severe attacks, dizziness was present so that walking was uncertain. About a month later the remaining members of the family were affected, the attacks varying in intensity, as in the father. The attacks rarely occurred during the early morning, but usually during the afternoon or evening, and they never occurred during sleep. With the onset of cold weather the attacks disappeared.

In the second family (Sch), about the middle of July, 1924, the father first noted that at times during his work he could not keep his eyes open and things blurred. Gradually these attacks became more severe and of longer duration. The two sons shortly complained of the same symptoms, but because of their moving away from the district could not be observed.

The examination during the interval period showed in the family L that both father and mother had a slight ptosis, and convergence was impaired in the mother and son. In the family Sch, there were no abnormal findings during this period. The father of the family Sch was observed several times during an attack. The outstanding finding was the ptosis which was so marked that with most forceful attempts the lids could not be raised. Vision was not disturbed, but convergence was entirely absent. The remaining eye movements were unimpaired. The other members were also observed in light attacks with essentially the same findings. No severe attacks were observed.

In seven patients short attacks of ptosis of the lids, convergence paralysis, weakness of the neck and mouth muscles, dizziness and visual disturbances were noted in the summer and fall of 1924. The disease appeared in the form of house epidemics. The findings presented by this group of patients is essentially like those described by Gerlier and Kubisagari. Thus far no anatomic cause has been determined for this affection. During attacks a leukocytosis was noted in several cases, which rapidly disappeared with the subsidence of the attack.

The relationship of Gerlier's syndrome to myasthenia gravis has been suggested, but there is only a superficial similarity. In myasthenia gravis the attacks are never so short. Other muscle groups are affected, and the condition does not occur in epidemics. Encephalitis has also been suggested as a possible etiologic factor, but this seems unlikely to the author. Ladame suggested that the condition was hysterical. The writer does not believe this to be true. The epidemics occurred in separated regions and in no way suggested a group hysteria.

The question of an infectious process from unhygienic conditions was investigated by the author, and while the house and barn were either adjoining or in close proximity, there was nothing to lead to the idea of marked uncleanliness.

No results were obtained by the author in inoculation tests, although cats, which appeared to be more sensitive to the affection, were not tried. Potassium iodid and solution of potassium arsenite (Fowler's solution) were used with uncertain results.

MOERSCH, Rochester, Minn.

A CONTRIBUTION TO THE PATHOLOGY OF LANDRY'S PARALYSIS. E. SAPAS, Jahrb. f. Psychiat. U. Neurol. **42**:151, 1923.

The question of the pathologic process in Landry's paralysis is still unsettled. One group of pathologists calls attention, both clinically and histo-anatomically, to the similarity between Landry's paralysis and poliomyelitis. They consider Landry's paralysis an acute form of poliomyelitis which does not differ from severe cases of infantile paralysis, since the latter often attacks the cells of the medulla and higher centers. These authors have been able to establish certain inflammatory changes in the cord which are almost similar to those of poliomyelitis. Among those holding this view are Spiller, Mönkeberg, Buzzard, Wickmann and others. Another but smaller group of pathologists holds, however, that there is no inflammatory process in Landry's paralysis. They consider the parenchymatous changes in the anterior horn cells to be toxic in nature, the toxins being of various kinds.

The author reports a case of typical Landry's paralysis which in his opinion demonstrates the fact that there is no given etiology in all the cases. The patient was a man, aged 42, who entered the hospital in October, 1920. He had

had typhus fever at the age of 13 and malaria at 40. A year previous to entrance in the hospital he had had a tumor removed from his left breast. About eleven months later, he began to have pain in his stomach and diarrhea, but did not vomit. A few days after this he had severe pains in both legs. Physical examination revealed flaccid paralysis of both lower extremities, with no active movements possible at the knee or hip joints. There was no Babinski or Oppenheim sign. The cremasteric reflexes were present, but the abdominal reflexes were absent on both sides. The patellar and Achilles reflexes were absent on both sides, and the biceps and triceps were both elicited. The patient could be lifted, but could not sit up voluntarily. In the upper extremities the movements at the shoulder were limited, while those at the elbows were practically lost. There was no muscular atrophy, no rigidity, no fibrillary twitchings and no disturbance in sensation. Lumbar puncture revealed seven cells, an increase in globulin, a negative Wassermann test and a negative culture. The patient died ten days after entrance from an intercurrent lung infection.

Necropsy was performed a few hours after death, and showed bronchopneumonia in both lower lobes, chronic emphysema, and hypertrophy of the right ventricle of the heart. Sections were cut at various levels in the cord and brain stem, and were stained with hematoxylin-eosin, van Gieson, toluidin blue, Spielmeyer's sheath stain, Mallory's anilin blue-gold orange stain, Haidenhein's hematoxylin-eosin, silver impregnation stain and Sudan stain.

Sections in the first and third sacral segments showed a bilaterally symmetrical disease of the ganglion cells of the anterior horn with the toluidin-blue stain. The disease process was localized chiefly in the ventrolateral and dorsolateral group of cells. The majority of the cells showed an acute swelling corresponding closely to the picture in primary cell disease—peripheral nucleus in the early stages, disappearance of the nuclei in the later stages and definite tigrolysis. Other cells showed a powder-like destruction of the tigroid bodies, with other cell changes as described above, and still others were less severely diseased as evidenced by a darker cytoplasm and nucleus. Besides these there were many cells with bizarre forms. The other cells of the anterior horn showed a relatively normal structure, and the author marvels that certain cells could show such marked evidence of disease, while others could remain apparently normal. The glia elements were essentially normal, except for a slight gliosis in the region of the dorsal root. Here also corpora amylacea were relatively frequent. With Bielschowsky's silver preparation a more or less pronounced fibrillary disease of almost all the cells was found, showing almost complete loss of neurofibrils. The cell axons showed no changes. With Spielmeyer's myelin sheath stain there was no loss of cells nor any evidence of focal disease. The white substance was intact throughout, except in the region of the diseased anterior horn cells, in which there was slight evidence of a degenerative change.

Examination of the fourth and fifth lumbar segments showed similar changes in the cells of the anterior horn except that in this region all the cells were affected and not only certain groups of cells. All degrees of cell change were found. Some cells showed merely a diminution in volume and size. No definite signs of an inflammatory lesion were demonstrable. Of many sections made from this region of the cord, a perivascular, mononuclear infiltration could be demonstrated in only two places, once about a vessel in the anterior sulcus, and once in the posterior horn. The cells were chiefly lymphocytes, with here and there a few plasma cells. Besides this there was a more or less outspoken increase in the endothelial elements of many of the vessels. Glia proliferation

was present in moderate degree. Silver preparations showed advanced disease of the netlike structure in the ganglion cells. The medullary sheath stain gave essentially normal findings. The Alzheimer IV stain showed an increase in certain glia cells with ameboid structures. The anilin-gold orange stain showed normal results.

In the upper thoracic cord, evidence of cell disease was much less marked, and only less severe cell changes were apparent, and the extreme cellular swelling which was found in the lower sections was entirely lacking.

The cervical cord showed cell changes similar to those in the sacral and lumbar segments, except that here fewer cells were affected, the difference being entirely quantitative.

In the medulla, the cells of the hypoglossal nucleus were for the most part normal, but a few showed evidence of acute swelling. The dorsal vagus nucleus revealed a diminution in size of many cells. Many cells of the nucleus ambiguus showed evidences of swelling, while the cells in the olive appeared smaller in size. The cells of the dorsal column nuclei were swollen. The facial nuclei gave no evidence of cell change. No evidences of inflammation were apparent anywhere in the medulla. The basal ganglia were normal in structure. The convolutions of the frontal cerebral cortex and the cerebellar cortex were normal both grossly and histologically.

ALPERS, Philadelphia.

A CASE OF BRAIN TUMOR IN THE ANT. RUDOLPH BRUN, Schweiz. Arch. f. Neurol. u. Psychiat. **16**:86, 1925.

In a nest of formica pratensis, a worker was observed which ran continuously in circles to the right. Obstacles were not avoided, and other ants were viciously attacked. The tarsus of the right anterior limb was slightly bent inward, and both antennae were held rigidly forward. On the following day, the right anterior limb and the right antenna were dragged along as though paretic. This behavior continued without interruption for six days and nights, when the specimen was placed in alcohol, then transferred to Carrazzi's acid-chloroform mixture to soften the chitin and embedded in paraffin. The specimen was brittle, and sections were prepared with difficulty. A few satisfactory ones were obtained which were stained with hematoxylin and eosin. A compact tumor occupied the left half of the superior cerebral ganglion; a marked destruction of both corpora pedunculata and the large somatochrome cells underlying it and the optic lobe were noted. The tumor was made up of very small granular cells which the author thought were probably of glial origin. A mycotic infection could not be excluded, and it was not possible to be absolutely certain of the histologic diagnosis.

The specimen was unusual, not only because of the tumor in an ant, but also because it afforded an opportunity of tracing certain well defined bundles by means of the resulting secondary degeneration in the brain of an insect for the first time. The tracts of particular interest were those of the ocellar glomeruli of Kenyon, or the tubercles of the central body of Viallanes. Thomson showed that these tracts are retroflex continuations of large posterior bundles proceeding from the corpora pedunculata. They divide under the corpus centrale, near the midline, into dorsal and ventral bundles which turn and proceed caudad. The ventral bundle goes to the fibrillar body of the protocerebrum, thence via the great commissure toward the ventral cerebral ganglion. Brun could not verify Thomson's conclusion that these tracts form a direct communication between the "cerebrum" and the abdominal ganglion chain,

constituting what might correspond to a pyramidal tract. The writer believes that this ventral tract ends in the ventral cerebral ganglion. The function of this tract is not known, but it may supply the mandibular nuclei. Thomson believes that the dorsal bundle ends in the corpus centrale, whereas Brun thinks it proceeds both directly and through secondary neurons to the bridge of Viallanes in the intercerebral region. He believes he has demonstrated definitely that this relationship is a crossed one.

In attempting to explain the various phenomena noted during life, he first discusses the running movements toward the uninjured side. Treviranus thought these depended on a disturbance of the sense organs on the corresponding side; however, removal of the eye, the statolithic apparatus, the sense of smell, and the antenna did not result in a deviation of the insect toward the corresponding side. Bethe and Loeb discovered that destruction of one dorsal cerebral ganglion altered the tonus in such a manner that the flexors became hypertonic and the extensors hypotonic. The rigid posture of both antennae at first, he explained on the basis of a bilateral apraxia due to commissural diaschisis. The paresis of the right foreleg was not easily explained; however, Brun believes that the forelegs share the function of the antennae as sense organs, and are not merely organs of locomotion as are the other legs which function more or less automatically.

The psychic disturbances which were so apparent have been noted in other insects after experimentation on the cerebral ganglia. Brun thinks they may be due in part to agnosia resulting from diaschisis. This would explain the indifference of these insects toward their colleagues; however, it would not explain the antisocial behavior. Some other factor, possibly a toxic one, must be present. These observations are, of course, only hypothetic.

WOLTMAN, Rochester, Minn.

MILIARY NECROSES AND ABSCESSES IN THE CEREBRAL CORTEX OF A PARETIC AND THEIR RELATION TO SPIROCHAETA PALLIDA. F. SCHOB, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:588 (March) 1925.

After Sträussler in 1906 described miliary necroses in the cortex of a paretic patient, similar descriptions were recorded by Grütter, and in particular by Hauptmann and Herschmann. These foci are of particular interest, as pointed out by the latter authors, since they stand in close relation to collections of spirochetes. The case here reported forms a further contribution to the knowledge of the histologic structure of these foci and of their relation to collections of spirochetes. It is a typical case of general paresis in a man aged 47, who had ideas of grandeur, dementia and positive clinical and laboratory signs of paresis. Microscopic examination of the brain at necropsy showed a definitely advanced paretic process in the cortex and meninges. Gummatous and endarteritic changes were not noticed. Besides the changes typical of paresis the cortex contained small necrotic foci, especially in the frontal areas, but less numerous in the occipital region, limited entirely to the cerebral cortex. Paretic changes were marked in the cerebellum and medulla. Three types of miliary necroses were seen: (1) foci in which the brain substance was changed into a homogeneous, necrotic mass which was filled with cellular elements or walled in, (2) abscess-like foci and (3) foci which were a mixture of necrotic and abscess-like processes.

The necrotic areas show noticeable histologic differences in various parts, but these represent different stages in the development of these foci. At

times these foci appear as circumscribed areas in the cortex at whose periphery ganglion and glia cells can be seen, though in a severely regressive state. Again, they may appear as homogeneous areas whose centers are very deeply stained and void of cellular elements. The majority of these areas show marked cellular reactions in their immediate vicinity in the form of a single or many-layered wall of cells surrounding the necrotic area on all sides or only on one side. Certain foci are made up of necrotic cellular elements.

The abscess-like areas showed a varied picture. At times they appeared as circumscribed areas in the cortex with a more or less thick collection of leukocytes, but without any visible disappearance of ganglion cells or glia cells. These foci were naturally not sharply limited. Other foci were visible in which all the cellular elements had disappeared. Still other areas contained a great many phagocytic cells, and more advanced areas showed a greater and greater predominance of these cells, with a disappearance of the leukocytes. The abscess-like foci were larger than the necrotic foci, their diameter being about twice as great.

A few foci showed a mixed structure, being composed of necrotic and abscess-like processes. These showed a necrotic area in the center more or less completely surrounded by a collection of leukocytes which sometimes formed a definite wall.

Of particular interest was the relation of spirochetes to these foci. In the necrotic areas, no spirochetes were found in the center, but with special spirochete stains a dark ring could be seen around this center which was formed of spirochetes. In other words, most of the spirochetes were at the periphery of these areas, though some were in the central homogeneous mass. In foci with a definite cell wall around them only few spirochetes were found. In the abscess-like foci the spirochetes were found between the cells; they were often seen in collections around the cells and often within the cells. Spirochetes were also found in the mixed foci.

The author looks on the necrosis as a primary process. Sträussler thinks they are a gummatous process, but Hauptmann and Herrschmann do not hold this view because necrosis is secondary in a gumma, while in this process they believe the necrosis is primary and the cellular reaction secondary to this. Hauptmann looks on these areas as a spirochetal colony which has arisen through multiplication of spirochetes with the brain substance as a medium. The spirochetes die in the center and multiply at the periphery, thus producing cellular reactions.

ALPERS, Philadelphia.

AN EXPERIMENTAL STUDY OF CELLULAR PROLIFERATION IN THE ANTERIOR PORTION OF THE SPINAL CORD OF AMBLYSTOMA. S. R. DETWILDER, J. Exper. Zool. **42**:333 (Aug. 5) 1925.

Replacing a portion of the embryonic spinal cord of *Amblystoma* (third, fourth and fifth segments, or fourth, fifth and sixth segments) by a unit of the cord comprising the first, second and third segments from another embryo brings about the following results:

(*a*) Cellular proliferation through the dorsal region of the transplanted segments of the cord is much higher in relation to that in the ventral region than in the intact anterior segments from a normal animal.

(*b*) Cellular proliferation through the ventral areas of the transplanted anterior segments is lower in relation to the dorsal (sensory) areas than in

these segments from a normal animal or from animals in which the anterior segments have been moved caudally, but preceded by an extraneous medulla.

(*c*) The dorsal regions of the intact anterior segments lying cephalad to the transplanted anterior ones (segments one and two or one, two and three), undergo cellular hyperplasia to such an extent that the dorsoventral relationships (size, shape and cell numbers) are entirely disproportional with the normal.

(*d*) The ventral regions of the anterior intact segments when followed by the transplanted anterior segments is only slightly affected.

The extent of proliferation characterizing the dorsal areas throughout the transplanted segments is due, for the most part, to an inherent capacity for extensive proliferation. It is probable that this region is somewhat affected by influences coming from the dorsal region of the contiguous intact segments. The results bear out in an experimental way the conclusions reached by Coghill, in that certain local regions of the central nervous system undergo differentiation and proliferation from causes which are hereditary in nature. The activities in these regions are entirely bound up with the development of the early reflex pattern. The experiments indicate that the dorsal regions of the anterior end of the cord retains its embryonic nature for a longer period of time than the ventral. The extensive cellular proliferation characterizing the dorsal portions of the intact anterior segments when followed by transplanted anterior segments suggests a strong dorsal influence passing cephalad from the graft. It is suggested that this influence may be commensurate with the extensive cephalic growth of the spinobulbar sensory pathways arising from these segments. Throughout the ventral regions of the anterior segments (normal and transplanted) cellular proliferation is relatively low as compared with the dorsal regions. When the anterior segments are transplanted caudally, but preceded by an extraneous medulla, the dorsoventral cellular relationships in the transplanted segments are essentially normal. This suggests that under normal conditions there is a bulbar influence which passes caudally and which affects the ventral regions of the anterior segments much more strongly than the dorsal.

The results warrant the interpretation that although cellular proliferation takes place independently of active function of one part of another, as shown by Coghill, in later stages different parts of the developing nervous system markedly affect morphogenetic processes in others. The evidence so far gathered from grafting experiments strongly favors the idea that this may be largely through the metabolic activity of growing and functioning neurones. The effect of injury of the cord upon the proliferation of cells as shown by Hooker seems to be of little consequence in determining the final number of cells characteristic of older larvae. The functional behavior of the larval animal may be entirely normal when marked structural changes from the normal exist within the central nervous system. This capacity for functional adaptation is a remarkable feature of the developing central nervous system.

WYMAN, Boston.

THE HISTOLOGY OF JUVENILE AMAUROTIC IDIOCY. J. G. GREENFIELD and GORDON HOLMES, Brain **48**:183, 1925.

Two cases are described, clinically, which occurred in the same generation in a family in which the parents came of pure English stock. Both boys were normal and in good health until 9 years of age, when gradual failing of vision

was noticed, followed later by epileptiform convulsions, mental deterioration and finally blindness and paralysis. Examination of the fundi revealed extensive macular and perimacular atrophy of the retinas. In these areas the retinas were grayish and had fine granular pigmentary deposits in them, while the optic disks revealed few or no changes. On the one who died at the age of 15, a complete histologic examination was made. There were no changes worthy of note in the internal organs or glands of internal secretion, except the suprarenals, in which there was intense fatty infiltration of the cells of the deeper layers of the cortex. The most striking gross abnormality in the brain was the small size and firmness of the cerebellum, which was about half its usual bulk, with marked sulci between the various lobes. Histologically the meninges and blood vessels were normal. The primary pathology was that of neuronic degeneration. The cells from all parts of the brain and cord were pear shaped, with a normal amount of Nissl granules toward the apical dendrite, the basal portion of the cell being distended with a granular lipoid substance to about twice its normal diameter. In some cells, as in the nuclei pontis, and the olivary and dentate nuclei, the change was more pronounced; the cells seemed to be completely filled with lipoid granules and to have lost all their Nissl bodies. In the cerebellum the actual changes in the nerve cells, and especially the Purkinje elements, were similar in nature to those affecting the cells of the rest of the nervous system, differing from them only in severity. At the base and along the course of the dendrites, rounded swellings filled with lipoid were frequently seen. The striking features were the complete absence of all cells of the granular layer, the diminution in number of the Purkinje cells, and the loss of basket fibers surrounding those cells of Purkinje which persisted, combined with a considerable increase of neuroglia in the position of the granular layer. The white matter of the cerebellum, as that of the cerebrum, was little affected, except for the frontopontile bundle. The most striking feature in the histologic changes in the retina was the degeneration of its outer layers, which was mild at the periphery and intense toward the macula. The earliest recognizable lesion was a breaking up of the rods and cones associated with a rarefaction and narrowing of the outer nuclear layer. The next stage was the replacement of the degenerated external layers of the retina by proliferating neuroglia, followed by a proliferation of the pigment epithelial cells and invasion of the degenerating retinal layers by them. The inner layers of the retina remained practically intact, except that the nerve cells of the ganglion layer were affected similarly to the cells of the central nervous system.

The authors reviewed the previous work done on this disease, as well as the findings in the infantile form, and advance the argument that the infantile and the juvenile types of amaurotic idiocy are manifestations of the same pathologic process, the histologic differences being mainly dependent on the mode of reaction of the nervous tissues and the incidence of the disease in special structures.

STACK, Milwaukee.

THE RELATION OF COMBINED SYSTEM DISEASE TO PERNICIOUS ANEMIA. LAUTERBACH, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:1 (March) 1925.

How common is combined system disease in pernicious anemia? Since the realization that this nervous disorder is a manifestation of Addison's anemia, it has been asserted that "next to tabes and multiple sclerosis the most common disease of the spinal cord" is this complication of primary anemia. Of 150

cases of pernicious anemia in the Mayo Clinic, Woltmann reports an incidence of combined system disease in 80.6 per cent. This coincides pretty closely with the figures of Minnich, who reports an incidence of 77 per cent. For the other 20 per cent. one may assume a rapid progression of the disease so that neurologic disorders could not develop, or else that spinal cord changes developed and could be demonstrated only at necropsy. The point which the author brings out is that spinal cord symptoms are present in the very early stages, with slight disorders of the blood pictures, and that these persist until death. In eighty-seven cases of pernicious anemia, she reports an incidence of combined system disease amounting to 38 per cent. She explains her low figures by the fact that the study is purely clinical, and that no pathologic material is included as in Woltmann's and Minnich's studies.

The classical picture of "funicular myelosis," as the author terms this neurologic complication of pernicious anemia, is manifested in four forms: (1) the spastic form, seldom seen as a pure type, (2) cases with spinal ataxia, pseudotabes in the true sense, (3) cases with outstanding cerebellar ataxia and (4) cases in which paresthesias predominate. Postmortem reports have emphasized the fact that changes in the spinal cord are apparent in cases of pernicious anemia with no cord symptoms or signs. Curschmann declares that there are few cases of primary anemia with no subjective or objective neurologic symptoms. This incongruity of clinical and pathologic findings has been ascribed by Nonne to a persistence of unmedullated axis cylinders.

Clinically, a positive Babinski sign may be the only sign of a lateral column disease. In other cases there may be only paresthesias or slight reflex changes. Typically, of course, one gets a combination or dorsal and lateral column symptoms—hypotonus and increased reflexes, eventually clonus or loss of reflexes and a Babinski sign. Differences in reflexes are particularly pathognomonic of column degeneration. These differences may be a loss on one side with a normal reflex on the other, or a retention of the patellar reflexes with a loss of the ankle reflexes. As to the blood picture, Hurst says that early in the disease the color index may be below 1, and there may be no normoblasts, whereas anisocytosis is present. He lays great stress on this sign. Schröder believes that in every case of combined system disease there is anemia, not always pernicious in type. Bramwell reports a case which for three years showed a typical picture of multiple sclerosis, until finally a typical blood picture of pernicious anemia was seen.

Views as to prognosis vary widely. Russell, Batten and Collier believe it characteristic of funicular myelosis that it is steadily progressive until death. Others hold this same view, and of course the prognosis is bad. In the author's experience, remissions are uncommon even with frequent remissions in the blood picture.

ALPERS, Philadelphia.

AMYOTROPHIC MENINGO-MYELITIS. J. P. MARTIN, Brain **48**:153, 1925.

The author applies this descriptive term to forms of spinal progressive muscular atrophy of syphilitic origin. In a case that had manifested itself by severe muscular wasting of the upper extremities, marked spasticity of the flexion type and only impairment of the vibratory sense in the legs, a complete histologic examination of the brain and cord was made which revealed: (1) a chronic leptomeningitis throughout the whole length of the cord and medulla, (2) a severe ependymitis, (3) an intense glial reaction toward both surfaces of the hemispheres, (4) intense degeneration of the white matter around the margin of the cord, especially of the anterolateral columns, the posterior

columns being but slightly affected, (5) degeneration of the anterior horn cells and cells of Clarke's column, almost absolute in the cervical enlargement but still definite throughout the remainder of the cord, (6) a moderate degree of arteritis affecting chiefly the intimal and adventitial coats of the vessels. Throughout the cord and meninges there was a moderate amount of perivascular infiltration with lymphocytes.

In another case in which the patient had presented severe atrophy of the upper extremities and neck with no other symptoms for more than twenty years, histologic examination revealed the same cellular destruction in the cervical cord, severe leptomeningitis and destruction of the fine fibers in the gray matter, but the marginal and tract degeneration was less severe. The author points out that in these cases we have all the elements of a diffuse meningomyelitis, and since one of the outstanding features in this condition is degeneration of the anterior horn cells, it merits the designation "amyotrophic meningo-myelitis." The pathologic findings, and also certain of the clinical and therapeutic characters of this condition make the author believe that the disease process begins in the spinal leptomeninges and that the destructive influence affects the cord mainly from the surface inward; the lymph channels probably aid its penetration to the central part of the cord, and would thus exert an early effect on the anterior horn cells.

From an analysis of sixty cases the main clinical features are given as follows: atrophy of the "atonic" type beginning (*a*) in the small muscles of the hand (*b*) in the shoulder muscles, or (*c*) in the muscles of the outer side of the leg; it may be unilateral or bilateral; its onset is not infrequently preceded by pain; spasticity of the lower limbs often occurs; bulbar symptoms are uncommon; vibration sense may be impaired, but superficial sensibility and muscle and joint sense have always been normal; there may be difficulties of sphincter control. Argyll Robertson pupils were present in only 28.5 per cent. of these cases. The Wassermann reaction was negative in the blood in one third of the cases; there was no sure case in this series in which it was negative in the cerebrospinal fluid before any antisyphilitic treatment was given. The author states that in his own experience and that recorded by others the progress of the disease has been arrested by antisyphilitic treatment.

STACK, Milwaukee.

THE INFLUENCE OF ORAL EROTISM ON CHARACTER FORMATION. KARL ABRAHAM, Internat. J. Psycho-Anal., July, 1925.

Abraham considers the problems of character formation from the unifying point of view afforded by infantile sexuality which involves the entire unconscious life of the mature human being.

Freud was the first to show that those elements of infantile sexuality which are excluded from participation in the sexual life of the adult undergo transformation into certain traits, and he formulated his well known "anal erotic" character distinguished by cleanliness, love of order and parsimony—the sources of which root down deeply to the earliest struggles over sphincteric adaptation.

Building on this, Abraham adds that the oral erogenous zone also is the source of character deviations as important as the anal and is perhaps of even greater importance in that it finds application in later life without undergoing repression and distortion. Nor can the aspects of one be considered without reference to the other.

The most primitive of human ways of obtaining pleasure is typified by the babe at the breast and is never completely abandoned. It persists under all kinds

of disguises during the whole of life and even experiences a reinforcement under particular circumstances.

This primitive pleasure is connected with taking in something from without to which is later added the retention or expulsion of bodily contents. If these are regarded as the prototypes of getting, giving or keeping, their great practical significance for the later social conduct of the individual can easily be seen.

The growth of the child involves a far-reaching renunciation of this oral pleasure. But every such renunciation takes place only when a "quid pro quo" is offered. Hence the child may leave this stage with difficulty or if disappointed or overindulged take pleasure in biting, which is the most primitive form of sadism and under the influence of an abnormally pronounced ambivalence leads to the predominance of hostile and jealous traits.

In fact, the vicissitudes of the libido in the oral phases may dominate the entire character, giving rise to certain well demarcated clinical types, depending on whether or not this period was one of gratification and whether the unconscious tendency is friendly or hostile. The purely oral character, according to Abraham, is bright, sociable, accessible to new ideas, importunate, restless, optimistic and generous.

In concluding, Abraham indicates the various combinations of oral, anal and genital manifestations in character and stresses the importance of not overestimating any one point of view, but of regarding character as a complex pattern into which are woven diverse strands only one of which he has here attempted to analyze into its constituent threads.

ROTHSCHILD, New York.

THE PATHOGENESIS OF SUB-ACUTE COMBINED DEGENERATION OF THE SPINAL CORD WITH SPECIAL REFERENCE TO ITS CONNECTION WITH ADDISON'S (PERNICIOUS) ANEMIA, ACHLORHYDRIA AND INTESTINAL INFECTION. ARTHUR F. HURST, Brain **48**:218, 1925.

This article is an elaboration on that published by the author and J. R. Bell in *Brain* in 1922. The author maintains that pernicious anemia and subacute combined degeneration of the cord are different manifestations of the same disease and practically always associated with achlorhydria. Thirty-six cases of Addison's anemia and twenty-four cases of subacute combined degeneration of the cord are reported, all of which showed complete achlorhydria by the fractional test meals. Of 579 cases of Addison's anemia reported by other authors, complete achlorhydria was found in 98.3 per cent., and it was present in all of the sixty-eight cases of subacute combined degeneration of the cord.

The author calls attention to the familial occurrence of Addison's anemia, and he collected records of thirteen cases, with one of his own, in which Addison's anemia in one or more members of a family was associated with achylia gastrica in others. A case of subacute combined degeneration of the spinal cord with Addison's anemia secondary to cancer of the stomach is reported clinically and pathologically, in which achlorhydria was present. Reference is likewise made to cases following gastrectomy and also gastroenterostomy in which the resulting achylia was a precursor of pernicious anemia, or subacute combined degeneration of the cord.

Oral sepsis consisting of streptococcic infection of the teeth, tonsils and tongue is believed by the author to play a strong part in the pathogenesis of these conditions. In twenty-eight cases of Addison's anemia and twelve of subacute combined degeneration, *Streptococcus longus* was found in the duodenal contents in 77.5 per cent. as compared with 20 per cent. of the 145 controls

used. In a great percentage of the cases the streptococcus had hemolytic properties. The author suggests that the streptococcic infection leads to the production of a hemotoxin and a neurotoxin, as Addison's anemia owes its specific hematologic characters to a toxin which acts directly on the bone marrow in addition to a hemolysin. The increase in the average size of the red corpuscles is stated to be the one essential characteristic feature of the blood picture, and in early cases may be the one distinguishing character of the blood picture.

As to treatment, early diagnosis followed by gradual removal of oral sepsis, continued use of hydrochloric acid and vaccine of the streptococcus obtained from the duodenum and teeth are recommended.

In a discussion of the paper, Dr. H. J. McBride, from the analysis of the gastric contents in fifty-five cases from the National Hospital, Queen's Square, in which fourteen patients had free hydrochloric acid on fractional test meal examination, stated that these facts could not support the gastro-intestinal intoxication theories, nor could they accept the facts that the absence of free hydrochloric acid was a constant predisposing factor. Others have reported similar findings.

STACK, Milwaukee.

TREATMENT OF MULTIPLE SCLEROSIS WITH SILVER CITRATE OINTMENT. SIEGFRIED FISHER, Med. Klin. **31**:733 (May) 1925.

Because of the spontaneous remissions in multiple sclerosis, the merits of any method of treatment must be judged cautiously. Also, hospitalization in itself often renders benefits not obtained in the home, and this makes it still more difficult to judge the merits of any therapy. The author, therefore, includes only those cases in which the patients pursue their regular occupations and largely follow their normal mode of living; and those cases in which the patients have improved under his therapy after having spent weeks and months in hospitals without change in their condition.

The author refers to fourteen cases of bona fide multiple sclerosis in which the patients had been treated by the Oppenheim therapy and collargol salve. By his method of treatment there was never an increase of symptoms, but at least a remission and frequently marked improvement.

There is no specific reaction in multiple sclerosis to indicate that the disease has abated, that is, there are no means by which one can be sure that it will not progress even when neurologic findings are negative. For this reason, even in view of negative neurologic findings, treatment is continued for a long time.

The following is the author's method of treatment:

Two grams of silver citrate ointment are applied for five days (salve to be fully absorbed). On the sixth day the patient bathes and rests on the seventh day. This is done four or five times. After a rest period of four weeks the same treatment is given, but 1 gm. of salve is used; again this is done four or five times. Then after one month's rest the treatment is again given. The process is continued accordingly for one year.

The case of a patient with spastic paraplegia of the legs and retrobulbar neuritis is cited, the disease having progressed for three fourths of a year. The patient had shown no improvement after many weeks of hospitalization. On institution of the foregoing therapy, the patient immediately walked better and soon was able to get about without a cane. The retrobulbar neuritis also improved.

A number of similar cases are cited.

The author believes that in silver citrate ointment he has found a therapy which will influence multiple sclerosis of not too long standing to a greater degree than any therapy which has heretofore been used. Similar gratifying results have been obtained in isolated cases of retrobulbar neuritis for which no other etiology has been found.

MOERSCH, Rochester, Minn.

THE PSYCHOSES: THEIR MECHANISMS AND ACCESSIBILITY TO INFLUENCE. ROBERT WALDER, Internat. J. Psycho-Anal., July, 1925.

Psychanalysis so far has thrown most light on the relations of the object libido which is directed toward the outside world. Its discoveries have been most effective in the therapy of the transference neuroses. Our knowledge of the ego instincts is relatively meager, and our power of influencing the mind does not extend to the psychoses since we lack here the dynamic transference energy through which neurotic fixations are overcome.

Walder restates the problem of the psychoses in analytical terminology and advances an hypothesis concerning the conditioning factors through which a psychosis comes about or is avoided in those borderline cases in which the phenomena of transition are readily observed, illustrating his theory by an interpretive discussion of an interesting schizoid personality.

Mental disease, as Walder conceives it, is not departure from a norm, nor does it represent a course of psychologic events which differs entirely from it. No mechanism has an inherent incompatibility with reality or an intrinsic real value.

But it is only the quantity of the libido, the material with which it is concerned and its relation to the whole psychic picture which does or does not confer a pathologic slant on the final result. Mental disease is incapacity to arrive at gratification of the desire for pleasure in any way that is actually possible or, restated, a turning away of libido from real objects and withdrawal into the ego. If this is all, we have a regressive psychosis such as catatonia. If following on the regression there is an attempt at restitution (a fresh turning of the libido to the outside world by the projection into that world of a part of the subject's mind), a restitutive type results.

The therapeutic task depends on the possibility of utilizing the libido which is flowing back into the ego and combining it with the existing sublimitations in a manner compatible with reality. The prognosis for intervention depends on whether in the accessible part of the personality germs of interests capable of development are found.

The problem is a union of narcissism and object libido. Walder believes the healthy remainder of the personality is the fulcrum of Archimedes by which the whole psychic cosmos can be moved and the narcissism changed to a form compatible with reality. By fostering insight and establishing communications where blockages have occurred, the task of the transference is simply to supply object libidinal positions, while the analyst stands for the patient's ego-ideal.

ROTHSCHILD, New York.

PATHOLOGY OF AMYOTROPHIC LATERAL SCLEROSIS. INASABURO NAITO, Jahrb. f. Psychiat. u. Neurol. **42**:90, 1922.

The exact nature of the process in amyotrophic lateral sclerosis is unsettled even at the present time. Marburg states that the nuclear amyotrophies and amyotrophic lateral sclerosis are degenerative inflammatory processes presumably on a toxic basis. The French, however, look on it as a poliomyelitis.

The process does in fact resemble the latter disease, as it attacks the motor nuclei and fibers predominantly. It is of importance to determine the primary seat of the process, and this is often the cervical cord and the medulla immediately adjacent to it. All the cases hitherto described have shown the result of a lengthy process. The author gives the pathologic findings in two cases with an acute course.

The first case showed a normal pia mater. This agrees with the findings of Schröder and Jakob. The ganglion cells of the cortex did not appear normal. The small pyramidal cells were fewer in number, tigroid bodies were not normally developed, and netlike formations were visible in the plasma. The giant pyramidal cells, however, showed the most severe changes. These showed an increase in fat pigment, and the nuclei showed evidence of severe degeneration. Lysis was visible. The nerve fibers, especially the radiary and tangential fibers, were severely degenerated. There were few gitter cells. Fat was visible in these areas. The entire cortex showed glia increase. The small glia cells were very much increased. Isolated vessels showed perivascular infiltration, but this was by no means general. These showed plasma cells and small glia cells in the perivascular spaces. The author concludes that there is a severe parenchymatous degeneration which affects chiefly the deeper layers of the cortex and the large pyramidal cells. The entire process is inflammatory in nature as shown by the perivascular infiltration, and is almost similar to what one finds in poliomyelitis.

The changes in the spinal cord are similar to those in the cortex except that the entire process is much more severe. The motor cells showed severe degeneration, the pyramidal fibers were degenerated, and there was an increase in glia elements. The second case which the author describes is very much like the first except that no perivascular infiltration was demonstrable.

ALPERS, Philadelphia.

A CLINICAL STUDY OF SENSORY JACKSONIAN FITS. OTTO SITTIG, Brain **48**:233, 1925.

The author studied the attacks of paresthesia which may be regarded as sensory jacksonian fits in ten of his own cases and quoted widely from the work of others, especially that of Jackson. He described the spread of paresthesia in ten cases due to cortical lesions, and showed that the course of the paresthesia in sensory jacksonian fits does not always follow the order of the sensory points in the postcentral convolution. He therefore concludes that sensory jacksonian fits are not simply due to a spread of an excitation along the sensory centers. According to the author's findings, sensory jacksonian fits may be grouped into three classes according to the manner in which the paresthesia spreads. The first class contains cases in which the sequence of the parts of the body affected by the paresthesia correspond strictly to the arrangement of sensory points in the postcentral gyrus. Especially characteristic is the spread of the paresthesia from the thumb to the corner of the mouth, and its tendency to remain limited or to extend but slightly. The second class consists of cases in which the paresthesia spreads over the whole half of the body and in which the sequence does not correspond strictly to the order of cortical points but usually is in the order of face, neck, shoulder, arm, and fingers; or toes, legs, finger, arm and face. In the third class the course of the paresthesia may correspond to an axial type (pre-axial and post-axial type). The paresthesia spreads either along the radial or ulnar aspect

of the arm, or along the anterior or posterior side of the leg in the order of metameric innervation, and from this fact it is inferred that there must be in the brain a representation of the metameric or axial arrangement. As to the explanation of these phenomena, the author is in accord with Hughlings, Jackson, Sherrington, and Head. If the stimulus is weak, the paresthesia does not spread widely and does not correspond with the order of sensory points in the postcentral gyrus, whereas if the excitation extends, it follows certain functional patterns which must not be regarded as pre-ordained centers and pathways.

STACK, Milwaukee.

ABNORMAL EAR CONDITIONS IN PATIENTS WITH MENTAL DISEASES. G. B. M. FREE, Arch. Otolaryngol. **1**:539 (May) 1925.

One thousand patients with mental diseases were examined over a period of two years for disease of the ear which occurred as a sequence or complication of the abnormal state.

Congenital deformities are without significance, though the older writers all emphasize their importance. Hematoma aurus, formerly said to be a trophic disturbance, is undoubtedly due to trauma. It is less frequently seen in patients given modern humane care.

Impacted cerumen may cause false perceptions in the form of auditory illusions and hallucinations, and the patient may hear imaginary voices. A case of a child of 11 years is cited. He was considered feebleminded at school because of frequent assaults on his fellow pupils, whom he imagined were talking about him. He would hear people behind him curse him. Removal of cerumen caused a complete cessation of the boy's bad conduct. The left ear is involved twice as often as the right due to the lessened dexterity of the left hand. Epilepsy due to lessened cutaneous sensibility and general sluggishness show cerumen more frequently than other patients with mental disease.

Disease of the abdominal viscera may cause a patient to think he has a snake in his stomach. In the same way, auditory hallucinations may be due to some abnormal condition of the ear, such as otitis media and eustachian closure. Deafness tends to the development of a suspicious temperament and in extreme cases to delusions of persecution. Mastoiditis is rare. Only one patient required operation at the Danville State Hospital during the last five years. Tinnitus may be interpreted as electricity being forced into the head. A patient with chronic otitis media had the delusion that there was a cricket in his ear. As a result of such delusions, the patients frequently beat the head and ears or put foreign bodies in the external canals: paper, soap, hair, wood, cockroaches, beetles and coal have been found.

The author makes no mention of mental states, depending on a toxemia, the source of which was middle ear suppuration.

HUNTER, Philadelphia.

THE EFFECT OF THYRO-PARATHYROIDECTOMY AND PARATHYROIDECTOMY AT 75 DAYS OF AGE ON THE GROWTH OF THE BRAIN AND SPINAL CORD OF MALE AND FEMALE ALBINO CATS. FREDERICK S. HAMMETT, J. Comp. Neurol. **37**:15 (June) 1924.

This paper is one of a series (No. XX) reporting the investigation of the rôle of the thyroid apparatus in the growth of the central nervous system from the point of view of age and sex relationships.

It contains the observations and discussion of the effects of the lack of thyroid and parathyroid secretion on the growth and differentiation (water solids)

of the brain and spinal cord of male and female albino rats, during the age period from 75 to 150 days, together with a comparison of the data of similar observations on control rats observed during the growth period from 100 to 150 days of age.

The author comes to the following general conclusions:

1. Sex and age are factors of importance in the determination of the type and degree of response in growth of the brain and cord to thyroid and parathyroid deficiency.

2. The brain is more dependent on thyroid function for its normal quantitative differentiation than is the cord. This difference is interpreted primarily on the basis of the relative time of appearance of the spinal cord, thyroid gland, and brain (cerebral hemispheres) during the course of phylogenetic evolution. It is secondarily interpreted by the differences in relative dependence of cytoplasmic and lipoid metabolism on thyroid function.

3. The growth of the central nervous system is more resistant to the toxemia of parathyroid deficiency than is that of the body as a whole.

4. The growth of the spinal cord is more resistant to the disturbances of metabolism induced by thyroid and parathyroid deficiency than is that of the body as a whole.

AUGUST, Detroit.

HAEMANGEIOMA OF THE PIA MATER CAUSING COMPRESSION PARAPLEGIA. PERCY SARGENT, Brain **48**:259, 1925.

The author reviews the cases published to date and adds four of his own verified by operation or necropsy. He states that the pathology of these cases is by no means clear, but that they resemble congenital nevi found elsewhere in the body and which may perhaps be regarded as of the same nature. In all cases there was nothing noteworthy as regards age, sex, or segmental level which might assist in differential diagnosis. The clinical history is regarded to be of great diagnostic value as the most striking feature was the remarkable manner in which the symptoms varied from time to time. In one of the author's cases, the symptoms varied from day to day, while in others improvement extended over a period of years. The author does not think that any real benefit was derived from operative interference in his cases, nor does he think the symptoms are wholly produced by mechanical pressure, but that they are more probably due to circulatory disturbances in the cord. Ligation of several of the veins while harmless was ineffective.

In connection with the foregoing cases an interesting case of arterial angioma (aneurysmal varix, plexiform angioma) is reported. A man, aged 44, suffered for two years with increasing weakness of the right arm, wasting of the muscles of the hand and pain in the shoulder. Soon afterward he developed a total paraplegia with profound sensory changes up to the first thoracic segment. It was noted that a large tortuous artery crossed the left supraclavicular fossa. An attempted laminectomy had to be abandoned because of severe hemorrhage. The muscles and bones were permeated with numerous dilated, tortuous, thin-walled arteries. At necropsy the cord was found to be compressed at the seventh cervical level by an aneurysm (illustrated) containing a recent clot. A transverse section of this level showed that the vessels within the cord were not affected.

STACK, Milwaukee.

THE USE OF LIPIODAL IN THE LOCALIZATION OF SPINAL LESIONS. F. G. EBAUGH, Am. J. M. S. **169**:639 (June) 1925.

This paper is a report of three cases in which iodized oil was used for the localization of subarachnoid block. In the first case, the clinical findings localized a lesion in the dorsal cord, with sensory disturbances beginning at the first dorsal segment. The oil tended to localize at the fifth dorsal segment, although some reached the sacral region. At operation, a "diffuse adherent inflammatory exudate" was found at the level of the fourth to sixth dorsal vertebrae, about the cord.

The second case gave evidences of a cervical cord lesion, but there was no localization with iodized oil. Later developments proved the condition to be epidemic encephalitis with radiculitis and parkinsonismus.

In the third case, symptoms and clinical study gave rise to the diagnosis of tumor at the fifth to seventh dorsal segments. The first lumbar puncture showed the fluid to be tinged with yellow, and it was impossible to obtain fluid subsequently. Iodized oil localized a block at the fifth dorsal vertebra, and operation disclosed a neurofibroma 4 cm. in length at the seventh segment.

The author's comments take up the value of combined cistern and lumbar puncture in conjunction with the injection of the oil. In the third case, sensory symptoms corresponding to the seventh dorsal segment followed injection of oil into the cisterna—and persisted for twenty-four hours. Otherwise no definite reactions followed the use of this substance. The ultimate fate of the oil and the effect of its presence in the spinal canal was not determined. Its use in the early diagnosis of subarachnoid block is emphasized.

PATTEN, Philadelphia.

"CONSCIOUS" AND "UNCONSCIOUS" IN PSYCHOLOGY. JAMES DREVER, J. Abnorm. Psychol. **19**:327 (Jan.-March) 1925.

The unconscious is recognized as an attribute of dispositional elements of the personality. In this sense there is nothing to come into consciousness. This attribute may be the associative bond that unites two ideas, or it may be thought of as the factor which determines the direction of the suggestion power of an idea to which is due the second or suggested idea. There is no suggestion as to what the nature of these dispositional attributes may be. Perhaps they are hormonic.

THE MEASUREMENT OF FUNDAMENTAL CHARACTER TRAITS BY A NEW DIAGNOSTIC TEST. ROLAND C. TRAVIS, J. Abnorm. Psychol. **19**:400 (Jan.-March) 1925.

In this article another method of making more objective the study of personalities is well described. The modest claims of the author after what seems to have been a careful and thorough piece of work are well summarized in his conclusions. The more interesting conclusions are: (1) There is no correlation between the scores arrived at and those of intelligence tests; (2) concrete statements are obtained which indicate the mental conflicts and personality drives.

HAMILL, Chicago.

THE ASYMMETRY OF THE SMALL-EYED CONDITION IN "EYELESS" DROSOPHILA. JOHN D. GUTHRIE, J. Exper. Zool. **42**:307 (July 5) 1925.

A study was made of the condition known as "eyeless" in a stock of white eyeless *Drosophila*. The character is asymmetrical, some flies being completely

eyeless and others having one or two small eyes. Two completely eyeless flies were crossed, and from this cross selection was started in two directions, one to increase the percentage of small-eyed flies and the other to increase the percentage of eyeless flies. From one to fifteen cultures were raised during each generation. Reciprocal crosses were made between the two lines of selection at the end of the twelfth generation for the eyeless selection and at the end of the tenth generation for those with eyes. Pupae from various positions on the glass of the culture bottles were examined, and a number of flies were raised from aseptic parents on sterile mediums. The results justified the following conclusions: The small eyes in "eyeless" *Drosophila* are distributed among the individuals of a culture chiefly on the basis of chance. The character yields to selection. Reciprocal crosses between two lines of selection show neither matroclinous nor patroclinous effects. The character is slightly sex-influenced, more eyes developing among the males than among the females. The number of flies with eyes developing increases with the age of the culture. Bacteria or the pupa's position are not causes of the asymmetry.

WYMAN, Boston.

CEREBROSPINAL FEVER. K. LEWKOWICZ, Lancet **2**:487 (Sept. 6) 1924.

Lewkowicz states his belief that meningococci penetrate the ventriculosubarachnoid system via the choroid plexus and not through the meninges according to previous opinions. The blood stream infection is of brief duration, due to the early development of immunity and the low vitality of the meningococcus. Since it has been noted that whenever a certain part of the subarachnoid space is cut off from communication with the ventricles and thus ceases to be continuously infected, the meningococcus disappears and the inflammation assumes a serous character, the author believes this to be a proof that meningitis independent of the ventricles does not exist. In explaining the presence of purulent exudate in the subarachnoid space, Lewkowicz states that the pus cells are carried to the subarachnoid space by the current of spinal fluid and deposited there when the fluid is absorbed. The idea that in cerebrospinal fever the ventricles constitute the only essential site of the infection is of great importance in treatment, for if this be true, therapy to be of the most value must be exhibited within the ventricles and probably best as serotherapy. In conclusion, the author describes his method of making ventricular puncture.

POTTER, Akron.

CONTROL OF PAIN, THROUGH THE NASAL (SPHENOPALATINE) GANGLION: REPORT OF CASES OF HERPES ZOSTER OF THE OPHTHALMIC NERVE AND OF THE BRACHIAL PLEXUS. CHARLES LUDVEY DAVIS, Arch. Otolaryngol. **1**:642 (June) 1925.

The author reports two cases of herpes zoster. In one along the distribution of the frontal and supra-orbital nerves, blocking the sphenopalatine area by topical application of cocain gave no relief, but blocking the anterior ethmoidal nerves gave complete relief on three successive days. The relief in this case cannot be explained. Anesthesia of one distribution of the fifth nerve which was not involved gave relief of pain in another branch of the fifth nerve.

In herpes zoster along the sternocleidomastoid muscle, the shoulder and the upper arm, with intense pain, the nasal examination was negative, but blocking the sphenopalatine ganglion gave complete relief. The patient failed to return after the first treatment.

HUNTER, Philadelphia.

DEGENERATION AND REGENERATION OF THE LATERAL LINE ORGANS IN AMEIURUS NEBULOSUS (LES.). MARY CHAMBERS BROCKELBANK, J. Exper. Zool. **42**:293 (July 5) 1925.

The lateral line branch of the vagus nerve of catfishes was severed by making an incision just dorsal to the lateral line and directly below the edge of the dorsal fin or just below the midregion of the dorsal fin. Fishes were killed at intervals after the operation and serial sections of their lateral line canals were prepared for microscopic examination. Study of the sections showed that degeneration of the lateral line organ begins within four days after cutting the lateral line nerve, is far advanced after ten days, and may continue for at least thirty-five days. Regeneration of these organs is conspicuous at fifty-four days, has progressed further at sixty-five days, and is practically complete at 116 days. Nervous connections appear to be necessary to keep the lateral line organs intact.

WYMAN, Boston.

TESTS OF HEARING OF FIVE HUNDRED AVERAGE EARS BY THE AUDIOMETER. DANA W. DRURY, Arch. Otolaryngol. **1**:524 (May) 1925.

Five hundred normal persons were tested with the audiometer in order to standardize the technic. Half of the subjects were males. The average age was about 18 years. They were divided into ten equal groups as follows: medical students, nurses, trained and untrained telephone operators, public and private school children, blind children, college and music students. The blind children do not hear more acurately than normal children, but their coordination is quicker. Children in an outdoor private school made a better record than those in a state school. The glee club, trained telephone operators and athletes made high records, showing the effect of training and of good health on aural acuity. High notes are apparently more readily perceived than low notes. The upper limit of hearing varied considerably.

HUNTER, Philadelphia.

OBSERVATIONS ON POST-ENCEPHALITIC PSYCHOSIS. P. R. MCCOWAN, Lancet **1**: 277 (Feb. 7) 1925.

This author selected eight patients with characteristic history of epidemic (lethargic) encephalitis from a group of patients who had been certified as insane. He calls special attention to the fact that mental defectives or those with an heredity of insanity are more likely to show postencephalitic psychosis. He concludes: Certifiable insanity is rare as a sequel to epidemic (lethargic) encephalitis. Many psychoses following epidemic encephalitis may not be true sequelae, the encephalitis acting merely as a precipitating factor. Psychotic symptoms during the acute phase of encephalitis are toxic in origin and offer a much better prognosis than postencephalitic psychosis.

POTTER, Akron.

LATERAL SINUS THROMBOSIS WITHOUT ELEVATION OF TEMPERATURE. J. W. HOLDERMAN, Arch. Otolaryngol. **1**:488 (May) 1925.

The author has collected thirty-one cases from the literature, including his own. Low virulence of infection, an obliterating thrombus, an aseptic thrombus or a fistula in the sinus with drainage through the mastoid cavity may account for the lack of the usual temperature curve. The reports show an equal distribution of afebrile lateral sinus thrombosis as a complication of acute and chronic middle ear disease. Fourteen patients had sinus abscess; the remainder had firm or totally organized thrombi; eleven had headache. In most instances, if we find a thrombus completely organized and no temperature disturbance, ligation of the jugular vein and removal of the clot is not necessary.

HUNTER, Philadelphia.

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, April 16, 1925

H. M. Adler, M.D., *President, in the Chair*

Case Report and Demonstration of a Pathologic Specimen of the Brain.

Dr. Arthur W. Rogers.

My object in presenting the record of this case is twofold: First, there is an unusual grouping of symptoms of a common condition, and second, it was only after a necropsy that a definite diagnosis was possible.

Mrs. R., aged 54, came under my observation, Nov. 11, 1924. She had been married for thirty-three years, had had one child, who was living, and no miscarriage or still-birth. She had ceased to menstruate four years previously. The family history was unimportant, and her health had been excellent. She had been attending to her full household duties until about two and one-half months before I saw her, when without premonitory symptoms she lapsed into a state of unconsciousness which lasted about one hour. At this time, as there had been some vomiting, her family physician interpreted the attack as of gastric origin; her stomach was washed out, and a diagnosis of autointoxication was made. She was more or less delirious for twenty-four hours after the attack, and, though she could be roused and answered questions fairly correctly, she could not, or at least did not, originate any ideas. In a week the family regarded her as quite well, and though complaining of severe pain in the back of her head, she got up and resumed her household duties.

About ten days after this first attack she had a second one, when she was again unconscious for about one-half hour; this attack was followed by transient left-sided hemiplegia with incontinence of urine and feces. Though the paralysis did not last more than thirty minutes, she was kept in bed for about ten weeks, complaining during all that time of severe frontal and occipital headache; the incontinence continued at varying intervals for about two months. She was mentally clear during this time; mentation was slow and deliberate, and she was irascible.

At the time I first saw her, examination developed nothing of particular interest other than the fact that all deep reflexes were considerably exaggerated. There was, however, an appreciable lack of the power of application with distinct lapses of memory, particularly for recent events, and apparent lack of pride in personal appearance. Urine findings and blood pressure were about normal, though the radials were slightly palpable. There were no pathologic blood findings. The patient said that she had lost between 30 and 40 pounds (13.6 and 18.1 kg.) in weight since the first attack of unconsciousness. She was discharged greatly improved on Jan. 8, 1925.

On February 28, I was called to her home in an adjoining town to see her. The husband informed me that since her discharge she had been in excellent health, although somewhat forgetful, and she had occasionally exhibited urinary incontinence. At this time she was partially unconscious, but could be readily aroused, and would answer questions promptly and cor-

rectly. She was perfectly oriented. This attack had been ushered in by vomiting, and in my presence she had distinct projectile vomiting. She complained a great deal of temporal headache. There were no sensory or motor disturbance, and no other special neurologic findings. The temperature was normal.

I did not see her again until five days later, when I found that unconsciousness had deepened. There were no important neurologic findings at this time except the absence of the epigastric and abdominal reflexes. The temperature and pulse were normal; respiration was 30 and shallow. The systolic blood pressure was 160. During her stay with me and up to date, repeated urinalyses have demonstrated nothing out of the ordinary. At this time an ophthalmic examination, although unsatisfactory, demonstrated considerable congestion of the retinal blood vessels. The nurse in charge who had seen a great many epileptic seizures reported that in the early morning, prior to my second visit, she observed a condition suggesting a mild general epileptiform seizure. Because of indications of a cerebral tumor, spinal puncture was not made.

The patient's condition grew worse and stupor deepened. Dr. Ogden of Milwaukee, in consultation, was of the opinion that we were dealing with a cerebral tumor, and the patient was removed to a Milwaukee hospital.

The first examination there showed a systolic blood pressure of 190, and the urinalysis revealed albumin and granular casts. The patient lived six days after removal to the hospital, and only during the last thirty-six hours of life were any neurologic symptoms noted, namely, the absence of the plantar reflex on the left side, twitching of the legs and double choked disks.

Necropsy demonstrated that on the occasion of her first attack of unconsciousness there was undoubtedly a hemorrhage into the right anterior lobe of the brain; this later became organized and produced symptoms resembling those of a tumor. This case demonstrates an unusual grouping of symptoms growing out of a generalized arteriosclerosis evidenced by the condition of the blood vessels of the brain and the fibrosis of the kidneys.

DISCUSSION

DR. H. DOUGLAS SINGER: The microscopic work was apparently done on a very small area, and I wonder whether it would not be advisable to search further for a tumor mass. The symptoms certainly point more to a tumor than to an atheroma of the vessels.

DR. ROGERS: The condition of the blood vessels at the base of the brain and the condition of the kidneys, as well as the cerebral hemorrhage, suggested the diagnosis.

TREATMENT WITH TRYPARSAMIDE OF PATIENTS WITH GENERAL PARALYSIS IN INSTITUTIONS. DR. CHARLES F. READ and DR. HARRY PASKIND.

In a series of ninety male patients with general paralysis observed and treated from July, 1923, to February, 1924, at the Elgin State hospital, forty-two were treated with tryparsamide and mercuric salicylate alone. The patients in Group A received one course of eight doses at weekly intervals, in Group B two courses with a rest period of six weeks between them, and in Group C three courses with two rest periods intervening.

In Group A (fifteen cases) the blood Wassermann reaction in one was not obtained at the end of treatment. Seven showed no change, five became negative, one showed a modified reaction, and one was anticomplementary.

The spinal fluid Wassermann reaction in fourteen cases was unchanged; in one it was not obtained. The cells in eight cases became normal; in two cases there was no change, and in two they were decreased. The Lange test after treatment was typical in seven cases and modified in seven. The globulin showed no change in seven, was reduced in four, and became negatve in two. Body weight after treatment showed no change in one, a gain in ten and a loss in four. The physical status of the group as a whole was slightly improved. The mental status before treatment was poor in four, fair in eight and good in three. After treatment it was poor in two, fair in six and good in three. There were four remissions.

Eighteen patients received two "courses" (sixteen doses) of tryparsamide and mercury. In this group, B, the blood Wassermann reaction showed no change in nine, became negative in seven and was modified in two. The spinal fluid showed no change in fifteen and a modified reaction in three. The cell globulin and Lange test changes were about the same as in Group A after treatment. The mental status before treatment was poor in six, fair in seven and good in four, with one remission. After treatment it was poor in one, fair in ten, good in five, and there was a remission in two. One patient died in convulsions after treatment had been discontinued, when he seemed to be in very good mental and physical condition. One of the two patients with remissions in this group came to the hospital in very good mental condition and with insight, hence is not included in further statements concerning remissions obtained.

The patients in Group C, nine cases, received twenty-four hour treatments. In this group five blood Wassermann tests showed no change, one became negative and three were modified. There was no change in the spinal fluid Wassermann reactions. The data concerning cells, Lange and globulin reactions were essentially the same as those indicated in the other groups. The general physical status remained stationary. The mental status before treatment was poor in two, fair in five and good in two. After treatment it was poor in one, fair in none and good in five, with three remissions. The remissions amounted to 33.3 per cent. in this small group. All three patients were in fair mental and physical condition when treatment was started, and during the period of treatment they improved physically and entered into a remission mentally. Of these three, one has been returned to his friends, seems to be making an adjustment to home life, and has begun to work as a night watchman. Two are making an excellent institutional adjustment and are soon to be paroled.

In all, then, this group of forty-two patients can be credited with eight remissions — excluding one patient who entered in excellent mental condition following an acute confusional episode — a percentage of 19 as against from 3 to 8 per cent. without treatment. The mental status of the group as a whole was considerably elevated. Eleven patients were treated with tryparsamide after treatment with neo-arsphenamin (two cases) or after sulpharsphenamin (nine cases). Of these, six received two courses of eight doses each and the remainder one course. The blood Wassermann reaction at the last examination was negative in six of this group (over 50 per cent.), but a four plus reaction became negative in only two cases after tryparsamide was given, in two it went from two plus to negative, in one case from negative to two plus. The spinal fluid Wassermann reaction was not influenced by the tryparsamide.

In March, 1923, nine cases were dropped at the end of the second course of tryparsamide on account of changes in the ocular fundi. Of these nine

patients, four now have normal disks and four still show changes. One patient was away and could not be examined. Of the fifty-three patients, thirty-five were reexamined, and pallor of the disks was noted in twelve, or 22.6 per cent. of the patients of this group. Five occurred after the second course, or 26.3 per cent. of those so treated. It is possible that some of these apparent atrophies are transitory conditions.

These findings are much worse than those of other reporters. The examining ophthalmologist was asked to eliminate all suspicious cases in view of the fact that he could see the patients only at long intervals, and no doubt many were dropped who would otherwise have been continued under treatment.

TRYPARSAMIDE AND SULPHARSPHENAMIN IN THE TREATMENT OF NEUROSYPHILIS. DR. ROBERT P. PARSONS.

The original plan for this investigation at the U. S. Naval Hospital, Great Lakes, was to divide all the neurosyphilitic patients admitted to the hospital into two equal groups, one for treatment with sulpharsphenamin and one for treatment with tryparsamide, with an attempt to have in each group an equal representation of the various forms of neurosyphilis. We were soon obliged, however, to abandon this plan on account of the amazing clinical improvement noted in most of the patients with general paralysis who were receiving tryparsamide, while those receiving sulpharsphenamin showed no evidence that they were being benefited by the drug. All patients with general paralysis were therefore placed in the tryparsamide group. Opportunity also was afforded by other types of neurosyphilitic patients who remained in the hospital long enough for us to observe the results of tryparsamide therapy after a lapse of a two month rest period following two courses of sulpharsphenamin.

Forty patients with neurosyphilis were treated during the last eighteen months, but only twenty-nine of these received sufficient treatment and complete enough serologic study to warrant consideration for purposes of drawing conclusions. Thirteen were treated with tryparsamide and sixteen with sulpharsphenamin. This is admittedly a meager group from which to draw conclusions, but the results on a percentage basis are for the tryparsamide cases essentially the same as those reported by observers who had opportunity to treat much larger groups, and for the sulpharsphenamin cases are nearly the same, i. e., not quite so promising, as those reported by Stokes and Behn in their group of twenty-eight patients treated with sulpharsphenamin. Our technic with tryparsamide was identical with that used by Lorenz and Loevenhart, namely, eight weekly doses of 3 gm. each to the course, with a weekly dose of 1 grain (0.06 gm.) of mercuric salicylate. Our technic with sulpharsphenamin differed from that used by Stokes and Behn in that they gave the drug intramuscularly; we gave it intravenously. It is possible that this explains why our results with sulpharsphenamin were not quite so good as theirs.

The results obtained with sulpharsphenamin were disappointing in many respects, but distinctly gratifying in others. It was used at first in several cases of general paralysis but with no success either clinically or serologically. On the other hand, symptomatic relief and serologic improvement were obtained to a degree in the other classes of cases, which I have never seen approached with the use of other arsphenamins. The incidence of visual disturbances was on the whole about the same as in the tryparsamide series. Two patients complained of blurred vision, one of whom suffered a permanent marked diminution of vision in one eye after the second course of treatment. He did not suffer any further visual damage or visual field contraction during

two subsequent courses of tryparsamide. An interesting feature here was that the patients treated with sulpharsphenamin never complained of visual disturbances until nearing the completion of the course of treatment, while as is well known, about 90 per cent. of patients showing visual trouble after treatment with tryparsamide do so after the first few injections.

Briefly, an analysis of our sulpharsphenamin cases is as follows:

The cases included eleven of meningovascular syphilis, four of tabes and one of the tabetic type of general paralysis. Mental symptoms were present in four cases, and cleared up in three under treatment. Neurologic signs were present in fourteen cases, in not one of which there was any change in these signs following treatment; two patients who had been suffering from epileptic seizures have remained free from seizures, one over a period of eight months so far, and one for two months. Thirteen of the patients had subjective symptoms; four of these have been considerably relieved subjectively and six have been completely relieved. Ten of the sixteen cases showed improvement in the spinal fluid; in two it became entirely negative, one was unchanged, and two were worse.

Our thirteen tryparsamide cases included four of meningovascular syphilis, two of tabes, one of the tabetic type of general paralysis and six of general paralysis. Seven of the patients had mental symptoms; five of them became entirely free from them and are working at present. Of the two who showed no mental improvement, one, I believe, had also dementia praecox, the other was suffering from a so-called fulminating general paralysis, and was undergoing rapid physical emaciation and mental deterioration when he began treatment. He stopped losing weight, in fact he gained 20 pounds (9 kg.) during the first course of treatment, but lapsed into a vegetative existence, resembling very much an old deteriorated, shut-in catatonic dementia praecox condition minus the delusions. Eleven of the thirteen patients presented neurologic signs. In six of these there was definite improvement neurologically; all of the eleven had pupillary changes. In one case the pupils became normal. In that case treatment was started one year after infection; during that first year the patient had received three courses of neo-arsphenamin. His spinal fluid was typically that of general paralysis, although there were no mental symptoms. Subjectively, seven of nine patients who complained of symptoms were completely relieved. Serologically, four of thirteen patients became well, the other nine showing definite improvement. The one showing the least improvement was one with general paralysis who at the time of admission had strongly positive blood tests, strongly positive spinal fluid tests with 0.2 c.c., 18 cells, globulin markedly increased and the Lange curve, 5555543100. One year later after four courses of tryparsamide, the blood tests were negative, the fluid tests still strongly positive with 0.2 c.c., 12 cells, globulin still increased and the Lange curve, 3333310000. This patient had gained in weight, however, as did all others, and made a clinical recovery. In cases of this kind in which the clinical recovery is not accompanied by negative serology after extensive tryparsamide treatment Loevenhart has recommended one or two courses of neo-arsphenamin or arsphenamin.

None of the patients treated with tryparsamide complained of blurred vision and none suffered any diminution of vision, although three showed a moderate constriction of the visual fields after treatment. The results obtained with tryparsamide were striking throughout and in some cases highly spectacular. The results compared favorably with those obtained elsewhere.

DISCUSSION OF TREATMENT OF NEUROSYPHILIS

DR. PETER BASSOE: In patients with general paralysis who are able to stay at home and come for treatment at the hospital or office I think perhaps we get a higher percentage of improvement than is noted in the institutional cases, as would be expected. I cannot give figures tonight, but I know that I have used about 1,800 gm. of tryparsamide. I can think of four cases, in three of which there have been four courses of treatment. In one the cell count and the Lange test are negative. In one there is decided mental improvement. That patient was a man aged 50 or 51 when treatment was started three years ago. He first received Swift-Ellis treatment and improved greatly, but then he had a relapse and was given tryparsamide. This was followed by improvement; later he deteriorated physically, while improving mentally, and he died last Saturday. I have treated eight or nine patients in the dispensary and several have improved serologically, but I cannot see any mental improvement.

As to the danger of optic nerve involvement, I am not afraid of that any more, and I think that it will not be long before tryparsamide will be generally used in the treatment of optic atrophy of syphilitic origin. I know it has been done here. One of my patients, who has now negative serologic findings, had some changes in the optic nerve when treatment was begun. He has received four courses of treatment and has been repeatedly and carefully studied by Dr. E. V. L. Brown; the condition of the optic nerve has improved during his treatment. I had a letter from Dr. Sydney Schwab, who is also using tryparsamide in many cases of this type, and he thinks that it should be used. There will probably be a repetition of the experience with arsphenamin. We were warned not to use that in cases of optic nerve disease, but now every one uses it, and so it will probably be with tryparsamide.

DR. SYDNEY D. WILGUS: I want to ask Dr. Read if in his opinion all these patients should be treated, and if so, how. Is it true that three or four courses would require a little more than six months? Few patients would stay for six months to receive the treatment, and they certainly will not unless marked improvement is manifested at a rather early date. How soon might improvement be expected? I had one recent case in which I gave two treatments and had started on the third when the family became tired and took the patient away. That man was beginning to get insight, and I thought the results were pretty good. I was deeply disappointed when his people sent him to Elgin.

I have tried the treatment in about fifteen cases, but I have never been able to complete what I would consider a full course of treatment. In about twelve cases the relatives have refused to allow the treatment because they have been told about the optic nerve changes, and if treatment has been started they will not allow it to be continued.

DR. SIGMUND KRUMHOLZ: I am somewhat conservative about using tryparsamide because of the effects on the optic nerve. I have not used it at all, even at the County Hospital. The report of Dr. Read that out of fifty-three patients treated, thirty-four have fundus changes and twelve have had optic atrophy or changes in the optic disks, does not seem to me encouraging. Of these, seven occurred during the first course, five during the second and about one-third of the patients treated had optic changes, if I understood him correctly. This is a large percentage. If it were other than optic atrophy, I would not mind, but to have changes in the eye, some of which will be permanent, is enough for me not to use the drug.

DR. H. DOUGLAS SINGER: Have you a series of carefully examined untreated patients?

DR. KRUMHOLZ: No, I have not.

DR. H. DOUGLAS SINGER: I have seen one case recently, in which the man came under treatment originally for tabes. He was treated with tryparsamide, and following this began to develop definite mental symptoms. He has received one subsequent course, but his mental symptoms are increasing.

DR. CHARLES F. READ: I think that last year we presented our rather meager results with a little more enthusiasm than we have in reporting our present results with a larger number of cases. I believe we found that tryparsamide is not liable to produce as many intermissions as have been promised. Certainly we have not been able to obtain nearly the percentage of remissions obtained by early reporters. Whether this is due to the character of the cases we have to deal with or whether it is due to the criteria involved in determining our remissions, I do not know. Certainly we have given the drug in the prescribed manner, and there can be no question of the therapy. When we remember that we have secured only eight remissions in forty-two patients treated with tryparsamide and that of these only three are now out and at work—which is the ultimate decision concerning remissions—we do not appear to have accomplished a great deal so far as returning the patient to normal is concerned, but we must credit tryparsamide with greatly improving the physical status, elevating the mental status and the behavior reactions of the group as a whole. It has held these people up remarkably. It has made them easier to handle in the institutions and has made the relatives happy. We have not had so many bedridden patients. We have not secured so many negative Wassermann reactions with the spinal fluid. We have secured more negative reactions with the blood, but that does not mean much. The reaction may be negative one month and positive the next. The serologic changes, or improvement, did not seem to parallel the mental changes. One patient who gave the best serologic changes we have had is a stupid person with general paralysis now. His intelligence is good, and he has no delusions, but he sits all day in a chair with his hand over his face.

Our patients were examined by an ophthalmologist in active practice, presumably a good man. Dr. Paskind will discuss this phase of the question. I know Lillie of the Mayo Clinic considers this no more harmful than other arsenicals. Other drugs we have used have brought about favorable changes, but no more than we could expect in untreated cases. I know some New York physicians have recently reported on a large number of treated and untreated cases and the percentage of remissions is about the same, but they all comment on the improved physical condition and perhaps some improvement in the mental state.

As far as institutional treatment goes, our conclusion is that we cannot expect much. The patients treated have all been committed patients. Perhaps too much destruction of the cortex had already occurred. It takes about five months to give two courses, which is a rather long time. We shall continue to use this therapy, and I feel that the treatment must be checked carefully by an ophthalmologist. I would not agree to use tryparsamide as we do neo-arsphenamin. It is unreasonable to suppose that within three months patients who had a normal fundus in the beginning would show marked changes in that length of time.

DR. HARRY A. PASKIND: Our experience checks up with that of Lorenz. In March we dropped eight cases because they showed changes in the optic disk. This month they were reexamined and four showed improvement while the other

four still showed changes. The patients we have treated could be considered as controls. The first examination showed no changes in the disks; if it had, we should not have treated these patients. Evidently the changes occurred after the treatment was instituted. We have tried to follow out this treatment with the sulpharsphenamin, but could not do so because of lack of time on the part of the ophthalmologist. My impression is that less optic changes occur under treatment with sulpharsphenamin than under treatment with tryparsamide, and that much less occur in untreated cases. Often in treating patients who have general paralysis one comes in contact with another psychosis. Three of these cases looked very much like dementia praecox. Another factor is alcoholism; we often come in contact with deterioration from alcoholism, which tryparsamide cannot be expected to reach.

Dr. Robert P. Parsons: None of patients was a committed patient, and we all thought the cases were early cases. Quoting Lorenz, we often find constriction for blue but not much constriction of the visual fields for form, although Wood of Baltimore says just the opposite. We have not seen enough patients with visual changes to hake a choice between these findings.

The difference between our results and those of Dr. Read may be accounted for by differences in the technic. All of these cases became either negative or less positive after the first course, and all patients treated for a year have negative blood reactions. Some have a positive spinal fluid Wassermann reaction, but all have low cell counts; some are between ten and twenty, none higher than twenty. As to globulin, some are reduced and some are not. The cell count seems first to return to normal. My experience has been that the least result is obtained in the Lange gold curve. We often get a negative Wassermann reaction on the spinal fluid with 0.2 c.c. and a practically normal cell count, while in the first two tubes there has been precipitation and several that we would call three or four.

SOCIÉTÉ DE NEUROLOGIE DE PARIS

Sixth Annual International, Neurological Reunion, May 25 to 28, 1925

This was a special meeting to commemorate the centenary of the birth of Charcot and to celebrate the twenty-fifth anniversary of the founding of the Society of Neurology of Paris. Because of Charcot's great interest in amyotrophic lateral sclerosis and in migraine (having described the former and suffered from the later), these two diseases were chosen as the subjects for report and discussion.

On Monday, May 25, the reunion opened at the Charcot amphitheater of the Salpêtrière with the reading of reports on amyotrophic lateral sclerosis. Professor Vincenzo Neri considered the disease from the clinical point of view. In introducing the subject, he admitted that Charcot, who first described amyotrophic lateral sclerosis, had written the best description, and one which holds today. More than fifty years of observation had added but little to our knowledge. Briefly, the chronic form may be described as follows: atrophy of the hand, associated with muscular twitching; the onset may, however, be paraplegic or bulbar. In fact, each case presents a different picture. The essential is a combination of the symptoms of disturbance of the lower motor neuron with those of the upper motor neuron.

Then followed a detailed description of the disease, incorporating recent observations. Fibrillary twitchings are considered important. Motor disturbances of the central nervous system are numerous and variable, the pyramidal tract being especially subject to injury. The curious variability of symptoms, such as the diffusion of reflexes, is due to the simultaneous presence of central and peripheral neuron lesions in different combinations. The muscular spasms are the result of a "sort of epilepsy of the ventral horn." Subjective sensory symptoms are common, but objective sensory disturbances and trophic disturbances are rare. Some patients show a slight emotional instability, but severe mental symptoms are unknown.

The onset of the disease is insidious and usually slow, but once established it proceeds to a fatal termination in two or three years, although occasionally patients have survived for ten years. In differential diagnosis one must consider multiple sclerosis, cervical gliosis, cervical myelitis and tumor.

The etiology is unknown; the disease is probably due to a virus. Since it is never hereditary and appears in middle life it cannot be classified with the abiotrophies. Syphilis as a causal factor may be ruled out by the normal spinal fluid and the statistics.

The subject was clearly presented, and showed good judgment in the weighing of evidence. It is to be regretted that references were not given to the authors cited.

Drs. Bertrand and Van Bogaërt of the Salpêtrière discussed the pathologic anatomy of amyotrophic lateral sclerosis. They have been especially interested in the cerebral lesions. In nine brains they found cortical lesions mostly in the precentral and frontal areas, affecting the third and fifth laminae and thus falling in line with Vogt's theory of pathoclisis. There are also small areas in which cells of all laminae are destroyed, obliterating the cytoarchitecture. Both in this way and in fiber destruction the ascending frontal convolution (Fa) seems to be particularly affected. Lesions were also described in the cerebral white tracts, the basal ganglia and the brain stem. Lantern slides were used to illustrate the presentation.

The essential pathologic picture is a degeneration of the pyramidal tract and its association fibers; but this is not complete; the cortical lesions are not constant and cannot cause the bulbar and cord degenerations. The most plausible explanation is that the affection is primarily one which attacks the bulbospinal gray substance, and, being "transneuronal," is able to cross the synapse and cause degeneration of the central motor neuron.

In the discussion Leri said that one must recognize a syphilitic myopathy which may be confused with progressive muscular atrophy and with amyotrophic lateral sclerosis. He did not believe in the existence of "poliomyelitis anterior chronica" as an entity separable from diseases mentioned above. In pathologic studies he thought that great care must be taken not to include syphilitic material with amyotrophic lateral sclerosis; he felt that Bertrand had not been careful enough and had shown syphilitic specimens.

Foix and Chavany showed lantern slides and described two cases with chronic degeneration of the ventral horn cells, the Betz cells and the motor tracts. They believed that the process began in the gray matter of the cord and affected the upper neurons by retrograde degeneration after jumping the synapse. Catola described a case in which there were lesions of the blood vessels. Poussep presented two cases which occurred in the same house. Marinesco gave an interesting exposition of his work on the chemical pathology of nerve cells in amyotrophic lateral sclerosis.

On Tuesday, May 26, Professor Guillain made an address on the historical development of the society, naming the great men and their conspicuous contributions to neurology. Particularly happy was his tribute to Prof. Meigs who had served for so many years as secretary to the society.

Following this address, representatives from various countries presented short papers on different subjects. These papers were concise and gave an idea of the trend of research, but were too brief to be reviewed. For example, Marinesco spoke on a spinal form of hemiplegia; Arias gave a paper on Royle's operation to relieve spasticity; Winkler described cases of tumor of the fourth ventricle; Brouwer described his experimental study of the retina and its central representation; and Dusser de Barenne told of his investigation of the postrolandic cortex by means of strychninization.

In the afternoon there was a meeting of the Academy of Medicine at which Prof. Pierre Marie eulogized Charcot. In the evening there was an official reception at which the President of the Republic was present. This took place in the great amphitheater of the Sorbonne, and the platform presented a brilliant academic spectacle. Addresses telling of Charcot's achievements were given by several prominent French physicians and scholars; speeches of congratulation were made by three foreign representatives, all others presenting theirs in writing. On behalf of the United States, Dr. Allen Starr presented a written greeting, and for the American Neurological Association, Stanley Cobb presented the following: "The American Neurological Association begs to present to the Societe de Neurologie de Paris most cordial greetings on this important and memorable occasion of its Twenty-Fifth Anniversary and the Centenary of the Birth of Charcot. American Neurologists enthusiastically join in congratulating their colleagues of France on the great advances in Neurology made under the leadership of the French School, and wish especially to add their word of homage to that of all nations which praise the illustrious master Charcot whose name has been, and will continue to be an inspiration to all students of Medical Science."

On Wednesday, May 27, at 9 a. m., there was an official visit to the Charcot Clinic at the Hospital of the Salpêtrière. Following this the reports on migraine were read at the amphitheater.

Viggo Christiansen of Copenhagen gave the clinical part of the report. It was an able description of the malady, about 12,000 words in length. He emphasized the fact that migraine is paroxysmal and that the patients are perfectly healthy between the attacks. He believes that it is strongly inherited, especially the ophthalmic form, and that it behaves in inheritance as a mendelian dominant character. He considers that the disease develops on some constitutional basis, not related to endocrine dysfunction, but manifesting itself largely in symptoms related to the sympathetic nervous system. He does not think that the relation between migraine and epilepsy is significant, although the "epilepsie sensitive" of Charcot is a frequent complication of migraine, but this is not a true epilepsy. For treatment he recommends a lactovegetarian diet, a well regulated life with little emotional excitement, and for drugs bromid and phenobarbital. The bromid may be associated with arsenic. Of special interest is the author's success with nitroglycerin given over long periods between the attacks. He has never had success with organotherapy. The report throughout was orderly, clear and reasonable.

The next paper was a long report by Pasteur Vallery-Radot on "The Pathogenesis of the Migraines." Though somewhat one-sided, this report

courageously supported the contention that an attack of migraine is an anaphylactic phenomenon. The symptoms are due to vagosympathetic disturbances; "ophthalmoplegic migraine" is considered a different entity, with cerebral or meningeal lesions. After reviewing the history of migraine from Claude Bernard and Dubois-Reymond down to the present, he summed up the evidence and concluded that the symptoms arise from vasoconstriction due to excitation of the sympathetic nervous system.

The causes of this sympathetic excitation were then discussed, and anaphylactic shock was considered the principal one, although in the endocrine sphere the ovary and thyroid were believed to play a part, and digestive troubles, excessive emotions, heat and cold may be important factors. All the latter excitants, however, may be merely incidental, setting off the colloidoclastic phenomena which cause the sudden disturbance of the neurovegetative system; this in turn brings into evidence the symptoms. Thus the true basis of migraine is a "colloidoplasmatic instability." The paper was well written, scholarly and well presented. Like Christiansen, the author does not believe in an essential relationship between migraine and epilepsy. An excellent bibliography was appended.

In discussing these papers many intesting points were brought out. De Lapersonne described the pale optic disk with contraction of the central artery of the retina, and said that the central vision may be lost during an attack of migraine with hemianopia. De Massary mentioned the frequency with which eating chocolate precipitated attacks; he considered the phenomena of migraine analogous to peptone shock, both being hypervagotonic. Haguenau had injected sterile milk into the temporal artery twice a week with good therapeutic effect. Boschi had no results from milk injection, but with long continued injection treatment with epinephrin or horse serum he had relieved many patients. Meigs described the vasomotor symptoms associated with the attacks and argued that migraine must have its origin in disturbances of the sympathetic system. Leopold Levi did not agree with the reporters in their skepticism concerning endocrinopathy in migraine. He had helped many patients with 0.002 gm. of thyroid a day, and believed that there was an instability of the thyroid. Fournier had seen two cases of migraine associated with "myxedema frustre." Poussep has found suprasellar tumors associated with migraine, and abnormal roentgenograms of the sella were shown in many other migraine cases. Leri said that the roentgenograms in these cases could not be relied on. He showed skulls with hyperostoses and "ulcerations," and said that chronic meningitis was not rare. Schroeder described his careful biochemical work, which shows ammonia dysregulation in both migraine and epilepsy.

In his final summary, Vallery-Radot said that his theory of etiology was based on the colloidoclastic hypothesis, and was, therefore, anaphylactic. He thought many cases showed hypothyroidism. Experiments with cat's smooth muscle showed contraction in serum from patients with epilepsy, migraine and urticaria.

In the evening a great banquet was held at the Palais d'Orsay in honor of Charcot. The chairman was M. Hesse, minister of colonies, and speeches were made by representatives of various countries. Dr. Starr responded in French to the toast "the United States of America."

On Thursday, the 28th, there was a reception at the Hotel-de-Ville to the neurologists attending the centenary and to the "Congres des Medicins Alienistes et Neurologistes" which was also meeting in Paris.

STANLEY COBB, M.D.,

NEW YORK NEUROLOGICAL SOCIETY

The Four Hundred and Twenty-First Regular Meeting, June 2, 1925

I. Abrahamson, M.D., *President, in the Chair*

A Lantern Slide Review of Some Uncommon Neuropathologic Findings. Dr. Joseph H. Globus.

A large series of lantern slides was shown. A number of them illustrated some unusual features in a series of well-known pathologic conditions of the central nervous system, such as the various stages in the development of solitary or multiple tuberculomas, the morphologic differences between a tubercle and a gumma, the almost constant coexistence of a more or less marked diffuse meningo-encephalitic process in the so-called basilar tuberculous meningitis and the various phases in the development of syphilitic meningo-encephalitis.

In another group of slides various glial reactions were shown with particular reference to the various stages of "roset" formations in their relation to neuronophagia and focal mobilization of glial elements.

Finally, a number of slides illustrating unusual features, peculiar cell forms and glial changes in some uncommon diseases of the central nervous system were also shown. This group included tuberous sclerosis with localized tumor formation, juvenile paresis, amaurotic family idiocy and progressive subcortical encephalopathy, the latter a striking degenerative disease process limited to the white substance of the brain.

Alcoholic Injections into Nerve Tissues for the Relief of Pain. Dr. George I. Swetlow.

In the following series of cases pain was the dominating feature. The relief obtained by means of alcoholic injections into nerve tissues was so marked that its use as a therapeutic measure may be highly recommended and widely used.

Ranşon, in his original researches on peripheral nerves, showed that a sensory peripheral nerve is made up of both myelinated and unmyelinated fibers and that the unmyelinated fibers carry the pain-bearing influences (protopathic). Since these fibers are peripheral arms or projections of cells situated in the dorsal ganglia, it is evident theoretically that the destruction of these peripheral arms would interrupt the pathway of the afferent stimuli and thus prevent the influx of pain stimuli to the sphere of consciousness.

The unmyelinated axons take their origins from the small cells in the dorsal root ganglia, while the myelinated ones originate from the large cells. The introduction of alcohol into a nerve produces an axonal degeneration with involvement, mostly of the small cells. The injection of an 80 per cent. solution of alcohol not only destroys the sensory component, but also the motor component. The injection of a 60 per cent. solution, however, produces only slight transitory motor weakness. The axonal degeneration of the afferent fibers and ganglia is at the same time extensive. The pathologic change found in a nerve and its ganglion cells following the injection of alcohol is the same as though the nerve itself had been cut. The microscopic picture is also that of a Wallerian degeneration.

Patients with these conditions were examined: (1) laryngeal tuberculosis, (2) pleurisy, (3) several forms of headache, (4) tabes with gastric crisis, (5) cardiac disease and (6) pruritus ani.

Thirteen patients with far advanced pulmonary tuberculosis with agonizing laryngeal pain were treated by the injection of a 60 per cent. solution of alcohol

into the superior laryngeal nerve. Complete relief was obtained. The periods during which the patients were relieved ranged from nine days to ninety-six days up to the day of the reading of this paper.

The following is a summary of the findings in the cases reported:

1. The injection of alcohol into the superior laryngeal nerve for the relief of pain is anatomically and physiologically rational.

2. The injection of alcohol relieves pain for several weeks at least.

3. Difficulty in swallowing due to pain is often completely relieved.

4. Cases in which there is difficulty in swallowing which is not due to laryngeal pain are to be rejected in this treatment. One cannot expect results in this class of patients.

5. The procedure properly performed is free from danger, and is practically painless.

6. The injection can be repeated if necessary.

7. This method has a definite and important rôle in the treatment of pain in tuberculosis of the larynx.

Five cases are presented as a preliminary report of an apparently new application of alcoholic injections into nerve tissue for the control of severe thoracic pain caused by pleuritic involvement. A careful search of the literature for any reference to this method has revealed no record of its previous use.

The patients treated were suffering from far advanced pulmonary tuberculosis with pleural involvement. The pain was severe in character. The duration of their complaints varied from five weeks to five months. Following the paravertebral injection with alcohol they were relieved. The duration of relief ranged from thirty-five days to 107 days to date.

Two patients with severe headache due to involvement of the great occipital nerve were treated with injection of alcohol into these nerves. The neuralgia was due to a cervical spondylitis. The duration of relief extended over a period of from seven to twelve weeks to date.

One patient with agonizing gastric crises was treated by means of paravertebral injection of alcohol into the posterior roots of the fourth, sixth and eighth dorsal segments on the right side and the third, fifth to seventh on the left side. The patient had been in extreme agony for nineteen days. He has been completely relieved for a period of seven weeks.

One patient with cardiac pain due to aortitis and aortic insufficiency on a syphilitic basis was treated. The complaint concerning pain dated back two years. Following the paravertebral injection of alcohol into the roots of the fourth, six and seventh dorsal nerves, the patient was greatly relieved. For a period of seven weeks, to date, she has been comfortable. The result possibly indicates the course of the pain stimuli in angina pectoris.

One patient who had severe pruritus ani was treated with the injection of 75 c.c. of a 20 per cent. alcohol solution into the epidural space. The patient had been suffering intensely for three years and was relieved by no form of treatment. Following the alcoholic injection he did not complain concerning this symptom. For a period of eighteen days, to date, he has been relieved.

Summary.—A series of cases diverse in nature were presented in which alcoholic injections into nerves were made, in order to obtain relief from pain.

The cases in which thoracic and cardiac pain were relieved were presented merely as a preliminary report.

The alcoholic injection into nerve tissue for the relief of pain is recommended highly.

DISCUSSION

DR. HERMAN SCHWATT: There is nothing so troublesome as the relief of dysphagia in laryngeal tuberculosis. At the Montefiore Hospital we have a great many such cases, and for many years we have tried one method or another for the relief of this condition. We have tried alcoholic injections a number of times, and have dropped this procedure, and taken it up again. As a rule, we have succeeded in relieving about 30 per cent. of the patients. It seems to me, since we have had occasion to observe the remarkable results of Dr. Swetlow, that it is all a matter of technic. Alcohol injection is not a new thing, but Dr. Swetlow has evolved a technic which is far superior to the one used before.

I can fully substantiate his statements. The patients whom he has treated have had cases of advanced pulmonary and laryngeal tuberculosis. Some of them had eaten practically nothing for a period of days and even weeks. They had been slowly starving, and the injection of alcohol into the superior laryngeal nerve by the technic of Swetlow worked a remarkable change. Some of the patients who had not been able to swallow can take various kinds of acid, salty, solid and liquid diet. This treatment is not intended for the cure of laryngeal tuberculosis, but any one who has seen these patients suffer from this condition should be delighted that a technic has been perfected by which they can be relieved during their last days.

In regard to pleuritic pains, it is not a frequent symptom in pulmonary tuberculosis. However, there are some patients who do complain of severe pain. As in the laryngeal cases, I want to state that the results obtained are marvelous, and it is a method to be recommended.

DR. SIMON ROTHENBERG: I have been very much impressed with the splendid work of Dr. Swetlow, and his fine presentation. He is to be complimented on the accurate method and technic used. All of us come face to face with the problem of pain, and all of us know how helpless we are at times. For a number of years I have used alcohol injections in certain types of cases with a fair measure of success in relieving pain. I have had no experience with laryngeal cases and none with pleuritic pains. I have used alcoholic injections in single nerve involvements, as in cases in which there was occipital nerve pain and unilateral headache. In these conditions the results have been very good. I also have used alcohol injections paravertebrally in cases of herpes zoster, with fair results. The use of saline injections in this condition has at times given me equally marvelous results. Certain cases of course will withstand any kind of treatment. That may be due to bad technic, but it is known that we do not obtain as good results in some as in others. For a number of years I have used alcohol injections in pruritus ani. Within the last year I had one patient at the Jewish Hospital who was treated in various ways and who was operated on twice for partial resection of the rectum, without relief from her symptoms. Alcohol injections around the anus and rectum gave her prompt relief, and she has remained well for the past year. I have never used epidural injections of alcohol for this condition. I have used alcohol injections also in certain type of brachial neuritis, using the injection paravertebrally, and have obtained satisfactory results, especially during the acute attacks of pain. In sciatica I have used alcohol injections from time to time, but I cannot say that I have found it to be advantageous over the use of ordinary saline injections given epidurally. I have, therefore, abandoned the use of it in those cases. I have, however, been much impressed with Dr. Swetlow's suggestion to use alcohol injections in angina pectoris. I have myself been working along these lines

and planned to use it in the next case I should see. It occurred to me that instead of injecting the alcohol paravertebrally in cases of angina, one might make an injection into the stump of the cut sympathetic nerve and in that way secure a wider effect than by merely cutting the nerve itself. I think this work is only in its infancy, and I am sure that with such splendid work as that of Dr. Swetlow, we shall obtain good results for otherwise intractable conditions.

Dr. W. M. Kraus: Dr. Swetlow's results, as has been said, are stimulating and suggest much work, not alone in the direction which he has indicated, but in other lines as well.

I think that the problem of the anatomy of pain fibers in the sympathetic nervous system is as involved as any which exists. The work of Langley on cats, in particular, showed that the sympathetic motor fibers follow a devious pathway, and that a general peripheral nerve contains fibers from a great many spinal segments. It is difficult to say whether the sympathetic sensory fibers follow such a path or go directly into the spinal cord at the same level as the nonsympathetic fibers. If we try to trace the sensory fibers inward, we are unable to say whether the sensory fibers, for instance, off the tenth thoracic nerve end in the tenth spinal cord segment, or whether they are diffused through a number of segments. I think with that problem in mind, and with Dr. Swetlow's method, we may be able to obtain information about this matter.

I can share Dr. Swetlow's optimism about his method of approach from the paravertebral point, and also a certain pessimism as to our knowledge of the anatomic course.

There is another question which is not remote, that of central pain. One thinks of Head's idea in this connection. It is possible that Dr. Swetlow's method could very well be applied to cases of pain in diseases of the central nervous system. It would be interesting if in a case of Parkinson's disease with a severe pain in the arm Dr. Swetlow would attempt to block the pain far from the lesion by cutting off afferent impulses. I think it is possible, with Head's work and Dr. Swetlow's work in mind, to do a great service in many of these extremely painful and incurable neurologic conditions.

Dr. S. P. Schwartz: I should like to call attention to a peculiarity in cardiac arrhythmias that occurred in one of these cases following treatment. The patient presented a sino-auricular block, and during the time of the block, symptoms of distress were absent, whereas previous to treatment she complained of skipping and jumping of the heart beat when the irregularity was present. This is just an incident in the work of Dr. Swetlow, but it should be considered in the light of future work in angina pectoris.

Dr. Swetlow said that before the injection of alcohol the patient was able to walk only a block and a half, and that following this injection of alcohol she was able to walk ten blocks without complaining of any pain or symptoms. During the last few days she was beginning to become conscious again of the same type of irregularities of which she complained before the injection. It is possible that the injection of alcohol releases the patient's consciousness of pain from irregularities.

Dr. Swetlow has brought out one point on which cardiologists are not as yet agreed, and that is the question of the relation of precordial pain to various areas of hyperesthesia, especially as found on the posterior part of the chest. A further search for these posteriorly will undoubtedly bring out some interesting facts concerning pain that might be relieved by some such methods as those described.

DR. I. ABRAHAMSON: I called Dr. Swetlow's attention to an excerpt in *The Journal of the American Medical Association* in which a definite belt of hyperesthesia was found in cases of mitral stenosis, again suggesting a method of approach by means of alcoholic injections in cardiac pain of mitral origin.

DR. SWETLOW: With reference to local injection in pruritus ani, I wish to say that, in considering this case, we decided to treat the patient with epidural injection of alcohol for the following reasons: In injecting alcohol locally, one often produces abscess formation and necrosis, particularly at times when the needle goes through the rectal wall. In trying to obviate this, we decided to introduce the alcohol into the epidural space. We went slowly and gradually, and worked up to a 20 per cent. solution. This has absolutely no ill effects. We also injected alcohol into another patient who is suffering from multiple sclerosis. There were no rectal or vesical disturbances.

With reference to the alcoholic injections in cardiac pain and the operative interference with the sympathetic ganglion, I wish to repeat that there are at least six different operations. Every man doing this work claims some success. A worse pain is substituted for the original pain. The suggestion made by one of the speakers to remove the ganglion and then inject the alcohol into the stumps is not justified.

Dr. Kraus has suggested the use of this method in syringomyelia with pain. If the pain were a level pain possibly due to a serous meningitis about the roots, we might be able to do something in this particular case.

With reference to the case in which the electrocardiographic reports were given by Dr. Schwartz, I wish to observe that I purposely excluded the report for the following reason: It would be an unfair presentation to bring this in, because the patient had just one electrocardiographic examination before I made the injection. After the injection she had several. We did not know whether these irregularities occurred before. Also, I cannot conceive that a ganglion situated outside the pathway of pain from the heart could have any effects on the heart mechanism. Dr. Schwartz tells me that many patients may have irregularities and not be conscious of them. The signs of cardiac exhaustion are not so much pain as palpitation and dyspnea. Many patients with marked cardiac weakness have no pain, but they know of their trouble by their dyspnea and palpitation. We can use these symptoms to regulate the activities of the patients.

END-RESULTS IN NEUROSURGERY; IMPRESSIONS DURING DECADE 1913-1922. DR. WILLIAM SHARPE.

During the past two years, I have been reviewing the records and examining the patients on whom I operated during the decade of 1913 to January 1, 1923. The records of only 73 per cent. of these cases could be completed up to date, the greatest difficulty having been encountered in locating the traumatic ward patients and the ambulance cases. My impression of the end-results from the standpoint of the ability of the patient to earn a living and to be a useful member of the community has been discouraging; particularly is this true of those patients who have had lesions of the central nervous system, such as brain tumor, brain abscess, chronic brain injuries and internal hydrocephalus, whereas more encouraging results have been obtained in the operative treatment of trifacial neuralgia, lesions of the spinal cord, brachial plexus and peripheral nerves, external hydrocephalus in its milder forms, chronic brain injury of supracortical hemorrhage, and, then, possibly the most gratifying of all, the

treatment of acute brain injuries, both in the new-born and in adults. In search the literature for detailed reports regarding the end-results in neurosurgery, the writer was very much surprised not to find such reports on any large series of cases. He found merely reports of individual cases in detail, and for a period of only two or three years after operation. The grouping together of brain conditions under one heading and considering the operative mortality rather than the end-result from the standpoint of a normal person, cerebellopontile angle tumors of the auditory nerve and benign in character and the operative relief of trifacial neuralgia with an operative mortality of less than 2 per cent. and a permanent relief of the pain, can in no way be compared with the seriousness of cerebral gliomas, subcortical abscess formations and the various types of hydrocephalus and chronic brain injuries, from the standpoint of future normality—physical, mental and emotional.

Discouraging Field.—(1). Brain Tumors: Eighty-one per cent. have been malignant, and none of these patients is alive, having been operated on more than three and a half years ago. Even if the tumor is benign, normal brain tissue has not only been irreparably impaired, but must frequently have been damaged in the removal of such a tumor, so that the individual cannot be considered normal. The operative mortality has been 19 per cent.

(2) Brain Abscess (a Cortical and Subcortical Lesion): Operative mortality has been 72 per cent. Confusion in the mortality statistics in the literature is due to subdural abscess formations of localized meningitis so frequently complicating mastoid disease being considered brain abscess. In the latter mortality is justly low—a mere dural incision suffices—whereas the seriousness of cortical and subcortical abscesses cannot be overestimated, both as to life and to future normality.

(3) Internal Hydrocephalus: A complete blockage of the ventricles in the aqueduct or in the posterior foramina has not been treated successfully to the present date, the mortality being practically 100 per cent. if surgical procedures are repeated in the hope of securing adequate drainage; no satisfactory method of drainage or permanent removal of the obstructive lesion has been devised.

Encouraging Field.—(1) Operative Treatment of Trifacial Neuralgia: The mortality has been below 2 per cent., and the relief of pain is permanent.

(2) Spinal Cord Lesions: The diagnosis is earlier and the localization more accurate than in cerebral lesions, and the percentage of malignancy has been only 41 per cent. The operative mortality has been 9 per cent. Early exploratory decompressive laminectomies are advocated in selected traumatic lesions of the spinal cord unless it is definitely known that all of the spinal tracts have been irreparably damaged and therefore in a hopeless condition; the operative mortality has been 19 per cent.

(3) Lesions of the Peripheral Nerves: End-to-end anastomoses are most satisfactory, and the earlier after injury in the absence of infection, the better is the prognosis.

(4) Surgical Repair of Lesions of Brachial Plexus, particularly the type of Brachial Birth Palsy in Children: The cases in which operation was performed numbered 146, with a mortality of one. The best functional results were obtained when the anastomosis was made at 3 months of age, and under 6 months of age; the older the child after this age, the more pronounced are the muscle contractures and the brachial shortening and deformities. Not one child, however, has obtained a perfectly normal arm, as normal as the unaffected arm.

(5) Chronic Peripheral Facial Paralysis: Anastomosis of the central end of the ipsolateral hypoglossal nerve with the distal end of the facial nerve; thirty-two patients were operated on with no mortality. The end-result has been merely an improvement at most, never a complete recovery of function.

(6) Brain Injuries: (*a*) Acute: (1) In Adults: In a series of more than 1,000 acute brain injuries in adults, the expectant palliative treatment, aided by repeated lumbar punctures of spinal drainage in many cases and dehydration by salines, has sufficed in two thirds of the patients, and in only 30 per cent. was a cranial operation of subtemporal drainage indicated—only when the intracranial pressure remained increased above twice the normal. The total mortality has been 29 per cent., and if moribund cases (11 per cent.) are excluded (those dying within six hours after injury from shock, other internal injuries, etc.), the mortality is lowered to 18 per cent. The operative mortality was 39 per cent., naturally, the more seriously injured patients having been operated on: first, during the stage of severe initial shock (if the patient survives, he survives in spite of the operation); and second, during the terminal stage of medullary edema, when all such patients die, operation or no operation, and the operation merely hastens death. Necropsy findings usually show supracortical hemorrhage of varying amount in the sulci associated with cerebral edema.

(2) In the New-born: Free blood of varying amounts in the cerebral spinal fluid of the new-born occurred in 9 per cent. of 500 new-born babies, lumbar puncture having been performed as a routine irrespective of the type of labor, within from twenty-four to forty-eight hours after birth. Clinical signs of its presence were frequently lacking, the most common being stupor, refusal to nurse and muscular twitches of the fingers and either orbit. During the past ten years, of forty-six newly born babies having signs of severe intracranial hemorrhage examined in consultation within two weeks after birth, lumbar puncture was bloody in varying degrees in 87 per cent.; spinal drainage by repeated lumbar punctures and cranial drainage by modified subtemporal decompression were used with varying results. Operative and necropsy findings disclosed supracortical hemorrhage of varying degree as the common pathology.

(*b*) Chronic: (1) In Adults: The usual complaints were persistent headache, dizzy spells, personality changes and convulsive seizures in a small percentage. Only the ones having a marked increase of intracranial pressure (more than twice the normal) and not lowered by medical treatment, glandular therapy, etc., were eventually drained by the subtemporal route, the findings being wet, edematous brains under pressure, and along the supracortical veins in the sulci was a cloudy, whitish, new tissue formation reported pathologically as being the organization residue of unabsorbed supracortical hemorrhage. The condition of these selected patients definitely improved following a lowering of the increased itracranial pressure.

(2) In Children: The type designated as cerebral spastic paralysis, usually due to a hemorrhage at the time of birth. As in chronic brain injuries in adults, only those having a definite increase of the intracranial pressure were operated on, and the operative findings were practically the same as in adults—chronic cerebral edema due to a partial blockage in the absorption of the cerebrospinal fluid through the walls of the supracortical veins by the organization residue of a former layer of supracortical hemorrhage. The operative mortality has been 10+ per cent. Improvement has resulted in more than 80 per cent. of the cases operated on, but only an improvement in these chronic cases, never a cure, the

ideal time for the drainage being, as in adults, at the time of the acute condition after birth, when the blood can be drained in fluid form, and thus there is no resulting condition of chronic cerebral edema.

DISCUSSION

DR. W. M. KRAUS: What I can say about the surgery of the nerves is almost entirely based on experiences in the war. Dr. Sharpe's comments on brachial plexus injuries are extremely encouraging. Unfortunately, I have seen only two cases operated on since the war. Dr. Sharpe thinks that there is some, if not a complete, recovery of function. The cases which I have seen showed only a partial recovery. It would be interesting to hear from Dr. Sharpe the ten years' summary of his work and the end-results he has had in causalgia. In my experience, those cases are almost always refractory to any sort of treatment; and the reason is very much emphasized by what Dr. Swetlow spoke of during the early part of the evening, namely, that we do not know the pathway of the sensory fibers. It would be interesting to hear whether Dr. Sharpe has operated in these cases, and if so, how many patients have recovered and the method of operation. I agree with what Dr. Abrahamson said about the paper, that it would be impossible to discuss all of it unless we stayed many days. The war ended with a vast number of the problems of peripheral nerve surgery still unsolved. For example, nobody seemed to be entirely convinced of the exact significance of electrical reactions. Has Dr. Sharpe had this test performed, and do the end-results in his cases show that the electrical response after operation confirmed in any way what he found at operation?

DR. ABRAHAMSON: I was very much interested in what Dr. Sharpe said about the acute injuries of the cord, especially the cases with signs coming on very soon, indicative of a transverse myelitis. I have always felt that nobody could say positively that the cord was thoroughly crushed. All we know is that there are signs of an acute transverse myelitis. Muskens and others found signs of a complete transverse myelitis in extradural lesions. In some of the cases there is not even a fracture of the lamina. A bullet had passed near the cord, and a complete transverse myelitis took place. I saw a patient recently who was accidentally shot by a policeman, and the bullet passed near the cord. The cord on operation looked normal, and yet the patient presented a transverse myelitis. No fragments were found that pressed on the cord. Why not give the patient a chance in view of this uncertainty? Dr. Sharpe's work in brain surgery in cases of trauma at birth, which he has stressed so much, is well known to all. He has done this work for many years, and some of his results are excellent. In my practice I have seen patients on whom he has operated, with marked improvement.

DR. E. D. FRIEDMAN: I think Dr. Sharpe has been a bit pessimistic over the end-results in brain surgery. We shall have to admit that curative effects are noted chiefly in lesions of the motor cortex and the cerebellopontile angle. Deep-seated neoplasms, of course, offer nothing more than the possibility of decompression in order to save the vision. We have found, both at Bellevue and at Mt. Sinai Hospital, that our mortality rate has not been very high and the results of decompression fairly good. Some patients have lived for periods varying from three to ten years after operation, the tumor having been localized but found to be irremovable.

The prognosis in cases of brain abscess secondary to suppuration in the lung is, of course, extremely poor. Those, however, which are otitic in origin, we

feel can be helped by exploration, particularly if such exploration is made through a small trephine opening in the skull. I recall one case in our service at Mt. Sinai Hospital that presented the evidences of brain abscess following an attack of otitis media. The exploration was made in the way indicated above in order to forestall the possibility of wide infection of the subarachnoid space. The abscess was found and drained, and the patient was discharged perfectly well.

One must agree with the statement that the results of surgical interference in the case of cord tumor are very much better than in cerebral neoplasm, for the reasons which Dr. Sharpe has already mentioned. One must also agree with Dr. Sharpe in the advisability of decompressive explorations in cases of serious injury to the skull, particularly in those instances which present evidences of increased intracranial tension, either by manometric studies of the spinal fluid or by the presence of papilledema.

Dr. Sharpe: It is gratifying to hear Dr. Abrahamson speak as he does regarding the present attitude of neurologists concerning acute injuries of the spinal cord. In 1916, I presented a paper to the society on this subject, suggesting that when it is not known definitely that a transverse myelitis is present, it is advisable to perform an exploratory laminectomy, because it is during the acute stage that a transverse myelitis may be merely simulated by compression of bone or hemorrhage; therefore, the ideal time for the spinal decompression is during the acute stage and not months later, when it is months too late, and any improvement can only be slight. In 1916, I was severely criticized for this apparently radical attitude, but I am still of the opinion that unless it can be demonstrated that the spinal cord is irreparably contused or severed, as might be inferred by the fracture-dislocation of the spinal column being out of alinement to a marked degree, the patient should be given the benefit of an early laminectomy during the acute period, in the hope that the spinal cord is not irreparably damaged.

Dr. Friedman feels that I am unduly pessimistic regarding the end-results in neurosurgery. Naturally I am not speaking of the operative results from the standpoint of the operative mortality, which is just a low one, not more than 16 per cent., and especially with the improved technic and the use of local anesthesia. I am speaking of the end-results from the standpoint of a normal person—one able to earn his living and be a useful member of society; it is from this standpoint that the end-results of conditions of brain tumor, true brain abscess, internal hydrocephalus and chronic cerebral lesions are uniformly bad. Unfortunately, the literature on the end-results in neurosurgery in detail is practically nil.

The end-results of severance of the posterior spinal roots for the alleviation of pain and the resection of portions of the sympathetic system for the lessening of spasticity have been most discouraging, and it is only in selected cases that the foregoing procedures can even be considered. None of my patients on whom I resected the cervical sympathetic ganglionic chain have been improved objectively, although one patient did state that the arm could be moved with greater facility and with less tension than before the operation.

Book Reviews

SYNDROMES NEURO-ANEMIQUES. PIERRE MATHIEU, M.D. Pp. 172. Paris: Gaston Doin, 1925.

The full title of Mathieu's thesis is: "Clinical and Anatomical Study of the Neuro-Anemic Syndromes, Especially the Subacute Combined Degenerations of the Spinal Cord with Anemia." He bases his observations on seven cases studied personally and a considerable though not nearly comprehensive review of the literature. Clinical description and classification are the main objectives, although he admits that there are so many gradations and combinations that classification is difficult.

The description of well developed anemic posterolateral sclerosis, "Lichtheim's syndrome," is careful, detailed and accurate, a fine clinical document. His own cases have been studied thoroughly from the neurologic point of view, and practically all of the clinical signs and symptoms have been included. Involvement of the brain and of the peripheral nerves is touched on; indeed, one of his patients showed polyneuritis with anemia. He points out that remissions in anemia are sometimes though not often accompanied by improvement in the nervous symptoms, and that relapses are accompanied by great increase in the severity of the neurologic symptoms. He divides the disease into three periods: prodromal, florid and terminal.

There is no new material, and the author does not lay enough stress on the importance of the tuning fork test in diagnosis. The worst omission, however, is in the study of the gastric secretions. In no case of the seven is there a report on the gastric analysis, and there is no discussion of the relationship of achlorhydria to the disease. Indeed the word "achlorhydria" appears only once in the thesis, and then in parenthesis. Surely there has been sufficient work done by this time to warrant a study of the cases from this angle. The recent important English works on the subject are not even mentioned.

Mathieu believed that anemia from various causes may be associated with subacute combined degeneration, and accepts the opinion that both hematologic and neurologic symptoms are due to the same etiologic factor. In two of the necropsies reported by him carcinoma of the stomach was found. Focal infection is mentioned in a cursory manner, but not the importance of the intestinal flora. The section on the pathogenesis of the disease is the poorest part of the work.

The report of the anatomic findings in the central nervous system, based on the work of Bertrand and Ferraro, is good, and is accompanied by numerous photographs and drawings.

Even as it stands, the essay probably marks a distinct contribution to the knowledge of the disease in France. Neuro-anemic syndromes are apparently rare in that country, although, as Mathieu himself says, there are probably numerous cases that have been overlooked.

DER HYPERKINETISCHE SYMPTOMENKOMPLEX UND SEINE NOSOLOGISCHE STELLUNG. By KURT POHLISCH, Assistent der Psychiatrischen und Nervenklinik der Charité Berlin. Price, 6 marks, unbound. Pp. 92. S. Karger: Berlin, 1925.

This monograph represents the author's effort to establish a distinct nosologic entity with hyperkinesia as the predominating feature in the clinical picture. In contradistinction to the encephalitic hyperkinesias, the term hyperkinesia as employed in this contribution is defined as a "disorder of motility which is based on a psychosis and can neither be explained neurologically nor psychologically." The increased psychomotor activity assumes the same characteristics and runs the same course in different persons and during recurrences in the same person. It seems to have a predilection for the female sex. In practically every case cited, the patient's personality was found primarily affected in its affectivity, thinking processes and consciousness. In a considerable number of cases extracerebral toxic factors played an important rôle; this was almost always the case during the first attack. Subsequent attacks were usually autochthonous in origin, and would follow each other with a remarkably precise periodicity. None of the cases showed definite evidence of a specific germinal predisposition such as one encounters in dementia praecox and in manic-depressive insanity. Neither symptomatologically nor etiologically could the syndrome be made to fit precisely into the prevailing conception of manic-depressive insanity or into that of dementia praecox. Pohlisch, however, points out that no matter which nosologic position one is inclined to assign to the syndrome in question, one fact stands out prominently, and that is that the clinical picture and course of the syndrome is the same whether it occurs on the basis of a manic-depressive psychosis, a dementia praecox, an exogenous psychosis or independently of any of these (epilepsy, paresis).

In substantiation of his thesis the author concludes the monograph with a detailed report of 62 cases selected from 250 cases under observation at the Charité in Berlin during the last twelve years. These particular cases were chosen because they lent themselves to a precise scientific investigation from every angle.

Without wishing to detract any merit from the author's efforts, the reviewer can see nothing in this thesis that has not been well known for a long time. Every psychiatrist is constantly confronted with mental cases in which increased psychomotor activity is a prominent feature, but in which, owing to other factors, it is almost impossible to determine whether this increased psychomotor activity is merely a symptom of any of the well established psychoses, or whether it is to be regarded as a clinical entity "sui generis." As far as the reviewer can see, the statement by the author that "these hyperkinesias cannot be classified and are to be regarded as symptom complexes which bear some relation to all psychoses" does not shed any additional light on this perplexing problem.